

## **4. RESULTS - EVALUATION OF HEALTH EFFECTS**

### **4.1 DESCRIPTIVE STATISTICS**

#### **4.1.1 Demographic Statistics**

The design for this study involved sampling children from twelve communities, chosen to represent a wide range of ambient levels of the air pollutants of interest. The communities were chosen on the basis of their historical ambient levels of  $O_3$ ,  $PM_{10}$ ,  $NO_2$ , and acid, where acid is the sum of  $HCl$  and  $HNO_3$  on a mole basis. For some of the communities, no air quality data were available; therefore, estimates were made based on interpolation. The data were particularly sparse for  $PM_{10}$  and acid.

Since choosing the communities, we have collected measurements of pulmonary function, developed estimates of lifetime ambient exposure to  $O_3$ ,  $PM_{10}$ , and  $NO_2$  on the individual level, and information on a variety of potential confounders and effect modifiers such as housing characteristics, morbidity (e.g. asthma), and physical activity (time activity) data, ascertained through questionnaires. Table 4.1.1.1 gives the numbers of subjects for whom various kinds of data are available. The estimation of exposure to the various pollutants is variable in quality and is discussed extensively in Section 3.

Table 4.1.1.2 presents the age distribution by community. The distributions are similar among communities, and the age distributions reflect the plan to sample 50% of subjects from fourth grade, and 25% each from the seventh and the tenth grades. Table 4.1.1.3 presents distributions of ethnicity and sex by community. Subjects were allowed to identify themselves by more than one race; those who did so were classified as "mixed". Many of the subjects who classified themselves as "other" also characterized themselves as "Hispanic". Therefore, it seems that many of these subjects were Hispanics who chose not to identify themselves as "white". These were included in all analyses, and were assigned to the category "other". In every community, more subjects identified themselves as "white" than as any other race. In 9 of the 12 communities, whites were in the majority. Santa Maria was the only community that was predominantly Hispanic. Racial differences are controlled for in the statistical analysis by including in the analysis several race indicators corresponding to the categories shown in Table 4.1.1.3. Therefore, while it is inevitable that racial differences will vary from community to community, this should not present a difficulty in the analysis of the data or the interpretation of the results.

Table 4.1.1.4 gives the distribution of annual family income by community. Most subjects came from families of middle to high income; only in Lancaster and Santa Maria did more than 20% of the families report earnings of less than \$15,000 per year. In Santa Maria, 28% of the children were in this category.

Much information can be gained by studying differences between subjects who are lifelong residents of their communities and those who migrated in. Table 4.1.1.5 presents the percentage of subjects in each community who were born outside the community. Most subjects were born outside their current community of residence. Most subjects were born in California, and of those born outside California, most were born in the United States.

#### **4.1.2 Residential Statistics**

Tables 4.1.2.1, 4.1.2.2, and 4.1.2.3 give information about the types, ages, and sizes of residences lived in by subjects. Most subjects live in single family homes. In all communities but Long Beach and Lompoc, residences of newer (aged 30 years or less) construction predominate. The modal number of bedrooms is 3 in every community except Upland, where it is 4. The average number of bedrooms is between 3 and 4 in every community except Long Beach and Santa Maria, where the average is slightly less than 3.

Table 4.1.2.4 provides some miscellaneous data on factors potentially related to indoor pollution or to lung function. Gas stoves predominate in every community but Alpine. The vast majority of homes have microwave ovens, and carpet in the child's bedroom. The percentage of homes with air conditioning varies greatly from community to community as expected because some are cooler coastal and some are hotter inland communities.

Since gas stoves are a potential source of indoor pollution, e.g.  $\text{NO}_2$ , we examined the methods by which they are lit. Table 4.1.2.5 gives the results. A majority of stoves are lit electronically, but many use continuously burning pilot lights and a few use matches.

#### **4.1.3 Estimates of Exposure**

In the analyses of the association of air pollution with health effects, three exposure estimates are used; historical exposure, 1994 data collected by our study team and lifetime exposure estimates. Each will be defined.

Historical exposure - Existing air pollution data collected between 1986-90 is called historical exposure. This is the same information that was used to estimate exposures for community selection described in Sections 2 and 3. This assigns the same exposure to everyone living in one of the twelve communities. We have done some analyses based on this approach but consider the next two exposure estimates to be superior because they better represent variation in individual exposure.

1994 Data - One of the goals of Phase II was to set up monitoring to improve upon past epidemiologic studies. The extensive sampling described in Section 2 allows us to improve estimates of the current exposures in our selected communities and allows

us to measure additional pollutants with potential health consequences, such as  $PM_{2.5}$  and acid vapor. This approach, while unable to determine past exposures, provides accurate assessment of current ambient exposures because the data are being collected specifically to characterize the exposures of children in this study.

Lifetime exposure estimates - Lifetime exposure estimates were obtained by combining residential histories obtained via questionnaire with records or estimates of historical levels of ambient pollution from approximately 500 towns in California (see Section 3). For each month in the life of each subject, the exposure level for each pollutant was taken to be the ambient level of the pollutant in the town in which the subject lived in that month. The lifetime exposure estimate was obtained by averaging the monthly exposures over those months for which data were available. Lifetime exposures were obtained for peak (daily 1-hr max)  $O_3$ , and average daily  $O_3$ ,  $PM_{10}$ , and  $NO_2$ . Historical records for ambient acidity were not adequate to allow construction of individual lifetime exposures for acid (for a fuller discussion of this issue see Section 3.2.1).

Figures 4.1.3.1 to 4.1.3.3 are histograms showing the distributions of lifetime exposure to peak  $O_3$  among participants in each of the twelve communities. There is a large spread of exposures in most of the communities. The shapes of the histograms tend to be different for high  $O_3$  level communities than for communities with low levels. In high level communities, such as San Dimas, there is a bulge at the right hand side of the histogram, representing the lifelong residents who have large exposures. The migrants (those moving in) form a long left-hand tail. In low level communities, such as Santa Maria, the directions are reversed. The lifelong residents form a bulge on the left, and the migrants form a long right-hand tail.

Figures 4.1.3.4 to 4.1.3.6 show the distributions of lifetime exposure to average daily  $O_3$ , figures 4.1.3.7 to 4.1.3.9 show the distributions of lifetime exposure to  $PM_{10}$ , and figures 4.1.3.10 to 4.1.3.12 show the distributions of lifetime exposure to  $NO_2$ . In all cases, a wide spread of exposures is represented in most of the communities, with the shape of the distribution reflecting the ambient level in the community, as with peak  $O_3$ .

#### **4.1.4 Validation of Activity**

Assessment of time spent outdoors and physical activity are important components of exposure models for ambient air pollution, since levels of pollutants typically differ between indoors and outdoors, and since greater physical activity involves increases in breathing rate and volume inspired. Both of these factors result in greater doses of pollutants delivered to the lung. In our study, we are particularly interested in usual patterns of activity because time outdoors and physical activity are subject to a high degree of day to day variation. However, the assessment of usual patterns of activity is problematic -- there are no well validated means of acquiring this

information. The problem is more severe in children. Because the schools are already granting us as much time as they are willing to complete the pulmonary function testing, we are forced to use a survey instrument which can be administered in a brief period of time to children as young as 10 years of age with only minimal coaching from adults. In designing an approach, we chose to focus on the periods when the child is in school because all children in a class have recess at the same time and their time outdoors and general level of activity is therefore subject to much less variability.

A detailed description of the rationale and methods of development of the physical activity questionnaires for this study can be found in the Quality Assurance Plan for the Phase II Study, Part IV: Questionnaire Surveys. We provide a brief description below.

In the first year of data collection, we used a computer scannable instrument which asked about the time outdoors in the afternoons in the previous two weeks. To avoid taking additional class time, we instructed the youngest children to take the instrument home and have their parents assist them in filling them out. A large proportion of these questionnaires were not returned. We called parents of these children to ask them the survey questions. In addition to the expense of calling, parent's reports of children's activity was notoriously poor. We therefore decided to change the instrument to one which could be filled out easily by younger children in about 15 minutes of class time. As described in the QA report, we benefitted from work published since the design of the first questionnaire and redesigned the questionnaire to be a 7 day recall. We tested prototypes in two focus groups and then did test-retest reliability on three versions and chose the two best (which had similar reliability) to acquire information on spatial, temporal and physical activity. These were incorporated into our questionnaire.

Knowing that validation of physical activity questionnaires is problematic because of the lack of a gold standard, we attempted to validate the instrument against diary data. The population for the validation exercise was the approximately 200 children enrolled in the HEI funded study of asthmatic, wheezy, and healthy children (HEI Research Agreement #93-4). The validation data were collected in the late summer and early fall. Children completed a daily diary broken up into one hour intervals. For each hour the children were asked to complete a page of the diary. The diary was a small booklet that children were asked to carry in a "fanny pack" provided to encourage them to fill it out as the day progressed rather than doing it as a recall at the end of the day. A small pencil was provided. A sample page is shown below:

7:00 a.m. to 7:59 a.m.  
**ACTIVITIES (PUT MAIN ONE FIRST):**  
 \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

**WHERE WERE YOU DURING THIS TIME?**  
 \_\_ home \_\_ near home \_\_ school \_\_ other

**AMOUNT OF TIME OUTDOORS:**  
 \_\_ none \_\_ some \_\_ most \_\_ all

**AMOUNT OF TIME IN TRAVEL  
 (CAR,BUS,VAN, TRUCK,TRAIN OR  
 MOTORCYCLE):**  
 \_\_ none \_\_ some \_\_ most \_\_ all

**AMOUNT OF TIME VERY PHYSICALLY  
 ACTIVE:**  
 \_\_ none \_\_ some \_\_ most \_\_ all

**BREATHING PROBLEMS:**  
 \_\_ wheeze \_\_ trouble breathing

**MEDICATIONS YOU TOOK:**  
 \_\_ puffer/inhaler \_\_ pill (or liquid)

Children were visited by study personnel at home and trained to fill out this diary from Thursday evening until Monday evening. Thus we had 4 days of diary information on each child. Children were called several times during that period to remind them to complete the diaries. If they had forgotten to fill it in, they were asked to go back and recall what they had done during the hours left blank. When the study staff visited the children to pickup the diaries, they were asked to fill out a 7 day recall (the same instrument used in the main study) covering the days of overlap.

To measure the validity of the information regarding amount of time outdoors, we summarized, for each subject, the information on whether the subject was outdoors from the HEI diary by scoring 0 for each afternoon hour during which the subject reported the time spent outdoors as "none", 1 for "some", 2 for "most" and 3 for "all", and summing the scores across the hours which overlap with the 7 day recall. For each subject, we summarized the outdoor information on the time-activity questionnaire by scoring 0 for each of the days Friday, Saturday, Sunday and Monday that the subject reported the time spent outdoors during the afternoons as 0 for "none", 1 for "some", 2 for "half", 3 for "most" and 4 for "all" and summing the scores.

The Pearson product moment correlation coefficient between the two values (estimates at time outdoors based on data collected with HEI diary as compared with the 7-day recall questionnaire employed in this study) above was  $r=0.45$  ( $p < 0.001$ ). The Spearman rank correlation coefficient was  $r= 0.44$  ( $p < 0.001$ ) which is quite good for survey work such as this.

To measure the validity of the information regarding physical activity we summarized for each subject, the activity information on the questionnaire by computing the number of days (out of 4) during which the subject reported engaging in one or more physical activities listed on the questionnaire, except "walking", "hanging out with friends", or "other". We summarized the HEI diary information by computing the number of days during which the subject reported being "very physically active" at least some of the time during the hours that overlapped with the questionnaire. Since each of these values is highly discrete (taking on values 0,1,2,3, or 4) the correlation coefficient is not an appropriate measure of association. We present the full distribution of variables below:

Number of days out of 4 during which subject was very physically active

Questionnaire	Diary				Total
	0 or 1	2	3	4	
0 or 1	7	2	3	11	23
2	2	2	4	6	14
3	7	8	5	15	35
4	15	11	21	81	128
	31	23	33	113	200

From the above table, it can be seen that 95 out of 200 subjects (47.5%) report identical results on both instruments. Out of 200 subjects, 147 (73.5%) reported results which are within 1 category. Thus the degree to which the questionnaire results in substantial misclassification of subjects (relative to the diaries) across categories is quite low.

In general, validations of children's physical activity questionnaires based on some more objective measure (direct observation,  $VO_2$  max, accelerometer) have resulted in correlations in the range of 0.13 to 0.60 (Sallis, 1991). Our results are on the high end of this range, suggesting that the instrument we used is comparable in validity to those used in other studies of childhood activity.

## 4.2 COMMUNITY HEALTH COMPARISONS

Table 4.2.1 presents the mean lung function by community as a percentage of predicted lung function. The predicted lung function was computed for each subject with a

linear regression, using the variables chosen by the method described in Section 2.

An analysis of variance was performed, comparing the mean values of the residuals across communities. We found no statistically significant differences for any of the five measures of lung function, which indicates that there is no evidence of systematic differences in lung function across communities. We used logistic regression to determine if the proportion of subjects whose  $FEV_1$  was less than 80% of predicted was related to the community of residence. No statistically significant association was found.

Table 4.2.2 presents morbidity prevalence by town and grade. Since the number of 7th and of 10th graders in each community is only about 75, and the number of 4th graders is about 150, differences of a few subjects result in apparently large differences in prevalence. There are no statistically significant associations between prevalence in communities and levels of exposure to air pollutants.

### **4.3 PULMONARY FUNCTION**

#### **4.3.1 Pulmonary Function by Community**

We first studied the effects of exposure to air pollutants with a between-communities analysis. In this analysis, all the subjects in any given community were assigned the historical exposure (common community exposure), i.e., the average of the ambient levels during the years 1986--1990. All other variables, such as age, size and gender, were measured on the individual level, however.

Tables 4.3.1.1 to 4.3.1.5 show the results. This was the exposure information that formed the basis for our original community selection.  $PM_{10}$ ,  $NO_2$ , and acid are associated with decreases in Forced Vital Capacity (FVC). Peak Ozone and  $PM_{10}$  are associated with decreases in Peak Expiratory Flow Rate (PEFR) (Table 4.3.1.3), and peak  $O_3$  is associated with a decrease in Maximum Mid Expiratory Flow (MMEF) (Table 4.3.1.4). The coefficients are larger for acid because the coefficients are based on change per unit of exposure, and the range of exposure units is much smaller for acid.

To attempt to separate the effects of the pollutants in cases where more than one pollutant was significantly related to the outcome, we fit models analogous to those in Tables 4.3.1.2, 4.3.1.3, and 4.3.1.4 with all significant pollutants entered together. The results are shown in Table 4.3.1.6. In part, because of the high correlations between historical measures of the various pollutants, none of the results are statistically significant. The lack of significance does not necessarily imply that there are no significant effects of one or more of the pollutants.

The results described above are subject to bias from both measurement error, resulting in exposure misclassification, and community-level confounding.

#### **4.3.2 Pulmonary Function by Individually Constructed Lifetime Exposure Estimate**

##### **4.3.2.1 Forced Expiratory Volume in One Second (FEV<sub>1</sub>)**

Potential confounders were screened by the method described in Section 2. The variables entered into the model were Hispanic origin, asthma, pets, and gas stove. Because the effects of lifetime exposure, and of other important explanatory variables such as height and weight, may vary among different age groups and between sexes, the results were analyzed separately for each of the three grades and two sexes. The results are shown in Table 4.3.2.1. Comparing the within grades results suggests an increasing trend in the magnitude of the O<sub>3</sub> effect with age, although it does not achieve statistical significance ( $p$  for trend for average ozone = .36, one-sided). While none of the differences between grades are statistically significant, the estimated effects for 10th graders were more negative than for 4th or 7th grades. The results presented in Table 4.3.2.1. show a significantly greater effect in boys than in girls for ozone (peak and average) exposure ( $p < .01$ , two-sided). At the same time the physical activity data clearly demonstrate that boys spend considerably more time outdoors, and more time very physically active, than girls (e.g., two-sample  $t$  statistic = 8.7,  $p \approx 0$ , for the difference between boys and girls in the mean number of weekday afternoon hours spent outdoors).

We also considered the effect of active smoking on FEV<sub>1</sub>. We defined as active smokers those subjects reporting having smoked four or more cigarettes during the week preceding the pulmonary function test. Active smoking was associated with an FEV<sub>1</sub> that on average was 283 ml larger ( $p \approx 0$ ). This will be discussed later (see Section 4.9).

##### **4.3.2.2 Forced Vital Capacity (FVC)**

Potential confounders were screened by the method described in Section 2. The variables entered into the model were Hispanic origin, and gas stove. Table 4.3.2.2.1 presents the effects of exposures estimated from this model. Peak O<sub>3</sub>, PM<sub>10</sub> and NO<sub>2</sub> have negative associations that are not statistically significant overall. We repeated the analysis separately for each of the three grades and two sexes. The results are shown in Table 4.3.2.2.2. As with FEV<sub>1</sub>, comparing the within grades results suggests that the magnitudes of all pollutant effects are greatest in 10th graders, although the differences do not achieve statistical significance. For peak O<sub>3</sub>, the estimated effect approaches, but does not attain statistical significance for boys.



#### **4.3.2.3 Peak Expiratory Flow Rate (PEFR)**

Potential confounders were screened by the method described in Section 2. The variables entered into the model were Hispanic origin, exercise just before the pulmonary function test, asthma, pets, pests, and exposure to passive smoke. Table 4.3.2.3. presents the effects of exposure estimated from this model. For all children combined, both peak O<sub>3</sub> and average O<sub>3</sub> show statistically significant negative associations. Average NO<sub>2</sub> has a statistically significant positive association.

We repeated the analysis separately for each of the three grades and two sexes (Table 4.3.2.3). There is no evidence of an increasing trend in the magnitude of the peak O<sub>3</sub> effect with age. Average O<sub>3</sub> tends to be associated with decreased PEFR in all three grades and in both sexes. Results for PM<sub>10</sub> and NO<sub>2</sub> are difficult to interpret at this point. The effect of PM<sub>10</sub> on 10th graders is significantly negative. Exposure to NO<sub>2</sub> is significantly associated with increased PEFR in 7th graders but not in 4th or 10th graders. Exposure to NO<sub>2</sub> is significantly associated with increased PEFR in girls, but not in boys. The reasons for these seemingly anomalous results are explored further in Section 4.3.5. The longitudinal study should provide further clarification.

#### **4.3.2.4 Maximum-Mid-Expiratory Flow Rate (MMEF)**

Potential confounders were screened by the method described in Section 2. The variables entered into the model were asthma, hay fever, and exposure to passive smoke. Table 4.3.2.4. presents the effects of exposure estimated from this model. Average O<sub>3</sub> has a significant negative association, NO<sub>2</sub> has a significant positive association. We repeated the analysis separately for each of the three grades and two sexes. Exposure to PM<sub>10</sub> is associated with increased MMEF in 7th graders. As with PEFR, exposure to NO<sub>2</sub> is significantly associated with increased MMEF in 7th graders but not in 4th or 10th graders, and with girls but not with boys (see Section 4.3.5 for further interpretation). Average O<sub>3</sub> tends to be associated with decreased MMEF in all three grades with a downward gradient by grade. Peak O<sub>3</sub> is associated with decreased MMEF in boys.

#### **4.3.2.5 Forced Expiratory Flow at 25% of FVC (FEF<sub>25</sub>)**

Potential confounders were screened by the method described in Section 2. The variables entered into the model were asthma and hay fever. Exposure to NO<sub>2</sub> was significantly positively associated, and average O<sub>3</sub> was significantly negatively associated (Table 4.3.2.5.).

We repeated the analysis separately for each of the three grades and two sexes. Peak O<sub>3</sub> is negatively associated in boys but not in girls. As with other measures of flow (PEFR and MMEF), there is a positive effect of NO<sub>2</sub> in 7th graders and in girls. Average O<sub>3</sub> tends to be associated with decreased FEF<sub>25</sub> in all three grades and both sexes.

#### **4.3.3 Pulmonary Function by Past Individually Constructed Exposure History: Migrants Only**

In high-exposure communities, migrants (defined as a person moving into the community) will tend to have less exposure to pollutants than lifelong residents. For this reason the findings of Section 4.3.2 could in principle be explained by confounding of air pollution exposure with migrant status. To address this issue, we fit the peak  $O_3$  models described in Section 4.3.2 to migrants only. The results, shown in Table 4.3.3.1, are quite similar to the results for all subjects taken together. This shows that the results of Section 4.3.2 are not due to confounding with migrant status.

We elected not to fit these individual-level models restricted to non-migrants, because among non-migrant children in a given age group in a community, there will be little variation in historical ambient exposure. Thus the accuracy of non-migrant specific estimates would be low. They would also be strongly confounded with age effects, since among lifelong residents of a community, cumulative exposure is very closely related to age.

#### **4.3.4 Pulmonary Function by Past Individually Constructed Exposure History, Adjusted for Physical Activity**

We screened the six physical activity variables described in Section 2.5 for entry into the models described in Section 4.3.2. None of the six were significant when entered individually into the models for  $FEV_1$ , MMEF, or  $FEF_{25}$ . One of the six (the number of hours outside during two summer weeks) was significant at the 15% level for both FVC and PEFR.

No interactions between these physical activity variables and exposure were statistically significant. Tables 4.3.4.1 and 4.3.4.2 show the effects on FVC and PEFR of exposure adjusted for physical/spatial activity. The association of peak and average  $O_3$  with FVC is stronger than without the adjustment (Table 4.3.2.2.1) although still not statistically significant. Estimated effects of exposure (average  $O_3$ ) on PEFR are slightly stronger with adjustment for physical activity (Table 4.3.2.3) than without (coefficient = -16.91 before and -17.76 after adjustment). Similar PEFR results were obtained for peak  $O_3$ .

#### **4.3.5 Multipollutant Models**

The apparently protective effects of  $NO_2$  noted above for PEFR, MMEF, and  $FEF_{25}$  were examined more closely in an attempt to explain them. Since these outcomes are highly correlated, we focused attention on MMEF, which showed the strongest association. We considered several possible explanations, including misspecification of the adjustment model, confounding by other exposures and differences in the characteristics of migrants to high vs low pollution communities.

To address the first possibility, we added several terms to the adjustment model beyond those listed in the footnote to Table 4.3.2.4., including grade, income (six categories), migrant status, and interactions of sex and grade with age, height, and weight, and the full set of race, technician, and spirometer codes (adjustment was made only for those that were selected in a stepwise fashion, as described in Section 2). These additional variables had very little influence on the regression coefficients or significance levels (also suggesting that family income reflecting socioeconomic status did not influence the results).

Next, we considered multipollutant models, in which we included peak ozone,  $PM_{10}$ , and  $NO_2$  in combination. The results were not qualitatively different, but the significance of both the deleterious ozone and apparently protective  $NO_2$  effects became stronger ( $p=.006$  and  $p=.007$  respectively). However, when we used average rather than peak ozone, this variable remained highly significant (as in the single pollutant model, table 4.3.2.4.), but the  $NO_2$  effect became nonsignificantly positive (slope=5.1,  $p=.17$ ). Addition of a quadratic effect of average ozone further reduced the  $NO_2$  effect (slope=3.5,  $p=.38$ ) and addition of peak ozone virtually eliminated the  $NO_2$  effect (slope=0.03,  $p=.99$ ). We interpret this to mean that average ozone is the important predictor of MMEF, not  $NO_2$ . Further investigation revealed that within all communities except Atascadero and Santa Maria, there are negative correlations between lifetime exposures to average ozone and  $NO_2$ , which explain why a model including only lifetime  $NO_2$  showed a positive association with MMEF.

Overall, lifelong residents do not have a mean MMEF different from migrants, nor is the effect of any of the three pollution variables significantly different between the two groups. However, the biggest difference occurs for  $NO_2$ : among migrants the positive effect of  $NO_2$  is 10.7 ml/sec/ppb compared with 5.8 ml/sec/ppb among permanent residents ( $p=.28$  for the difference).

The effect of  $NO_2$  also differs significantly between communities in the single pollutant model. The slope is significantly positive in Lake Arrowhead (25.3 ml/sec/ppb,  $p=.0085$ ) and Lancaster (28.4 ml/sec/ppb,  $p=.0001$ ) but not significantly different from zero in any other (it is nonsignificantly negative in Lompoc and Long Beach). However, there did not seem to be any consistent relationship between these slopes and the average pollution levels in these communities. The migrant effect also differed by community: lifelong Lancaster residents have significantly lower mean MMEF than migrants (mean difference -284 ml/sec,  $p=.02$ ), and Lake Arrowhead residents are also somewhat lower (mean difference -160 ml/sec,  $p=.18$ ); lifelong residents of Mira Loma have slightly better mean MMEF (mean difference 223 ml/sec,  $p=.07$ ); Lompoc and Long Beach are unremarkable, however.

Among permanent residents, there was no evidence of an effect of any of the three pollutants (unadjusted for community). For migrants, the estimated effects of the three pollutants are similar to those estimated above for all subjects combined: -6.3 ( $p=.006$ ) for ozone; 2.1 ( $p=.59$ ) for  $PM_{10}$ ; and 10.9 ( $p=.0049$ ) for  $NO_2$ . Thus, it appears to be the migrants alone, not the difference between migrants and nonmigrants that produces these effects. Grouping the communities into low, medium, and high for each pollutant on the basis of Table 3-13 (1986-90 values) revealed that the lifetime pollution effects appear to be confined to migrants to low pollution communities (for ozone) or to low and medium pollution communities (for  $NO_2$ ). Amongst migrants, the community-specific  $NO_2$  effects are almost identical to those given above, positive for Lake Arrowhead and Lancaster, nonsignificantly negative for Lompoc and Long Beach.

After adjustment for the effects of lifetime exposure to ozone,  $PM_{10}$ , and  $NO_2$ ; significant deleterious effects of ambient  $PM_{10}$  and  $NO_2$  were found in between-community comparisons. In multi-pollutant analyses, only the  $NO_2$  effect remained significant.

In summary, the apparently protective effect of  $NO_2$  could not be explained by other pollutants, by misspecification of the adjustment model, or by migrant status. However, it was quite variable between communities (as might be expected based on the smaller sample sizes) and appeared to be generally stronger amongst migrants to low pollution communities.

It seems most likely that the effect is mainly due to some misclassification of the ozone effect. Since the two pollutants have complex chemical interactions in the atmosphere, if there is a truly deleterious effect of ozone which is misclassified in a way that is correlated with  $NO_2$ , the latter variable might absorb some of ozone's real effect. In 10 of the 12 communities, there is a negative correlation between  $NO_2$  and  $O_3$ . Further insight into this possible explanation will have to await more detailed data on the measurement errors in the lifetime exposure variables that are being collected. If migrant status is part of the explanation, this will be clarified in Phase III, where longitudinal comparisons within subjects will be made.

#### 4.4 ANALYSIS OF 1994 PULMONARY FUNCTION DATA

We conducted some analyses of the relation of 1994  $FEV_1$  and FVC to estimated lifetime ambient exposure to air pollutants, using the pulmonary function results from the second year of testing. The results are based on those subjects who provided questionnaire information in 1993.

Table 4.4.1 presents the results for  $FEV_1$ . The 1994 results are similar to those from the 1993 data. Peak  $O_3$  and average  $O_3$  have effects that are less significant than in 1993.  $PM_{10}$  has a stronger positive effect in 1994 than in 1993, but it is not statistically significant.  $NO_2$  has no significant effect in either 1993 or 1994.

Table 4.4.2 presents results for FVC. As with  $FEV_1$ , the 1994 results are quite similar to those from 1993. Peak  $O_3$ ,  $NO_2$  and average  $O_3$  have negative effects on FVC that are not statistically significant.  $PM_{10}$  has a statistically nonsignificant positive effect in 1994; the effect in 1993 was nonsignificantly negative.

## **4.5 MORBIDITY ASCERTAINED BY QUESTIONNAIRE**

Morbidity was assessed in two ways. The first was by the annual questionnaire which acquired the medical history in 1993 including information on wheeziness, asthma, bronchitis, pneumonia and other respiratory diseases (see Section 2.3.1). The second was by absence monitoring in 1994 with a focus on respiratory illness (see Section 2.3.3). The results we report here relate to the first approach (annual questionnaire). The results of morbidity based on absence monitoring are reported in Section 4.6.

### **4.5.1 Morbidity by Community**

We studied the effects on morbidity (questionnaire based) of lifetime exposure to air pollutants with a between-communities analysis. In this analysis, all the subjects in any given community were assigned a common exposure. Other explanatory variables were measured on the individual level, however.

Tables 4.5.1.1 to 4.5.1.6 present the results of fitting a model in which exposure to each pollutant is assigned to be the ambient level during the years 1986 - 1990. This was the exposure information that formed the basis for our original community selection. No significant effects were found.

### **4.5.2 Morbidity by Past Individually Constructed Exposure History**

#### **4.5.2.1 Asthma**

We defined as cases of asthma all subjects who reported on the annual questionnaire more than one asthmatic episode in the previous 12 months as well as those subjects with exactly one asthmatic episode in the last 12 months who reported taking medication, losing sleep, or being hospitalized with the disease. For a sample of subjects who reported physician-diagnosed asthma, we validated the information by calling the physician. The diagnosis was consistently confirmed.

Potential confounders were screened by the procedure described in Section 2 and the variables entered into the model were hayfever, health insurance, more than five houseplants, pests, and carpet in the child's bedroom. Table 4.5.2.1.1 presents the effects of exposure estimated from this model. All pollutants are associated with small increased risk, with peak  $O_3$  approaching statistical significance.

To determine if effects of air pollution vary between the sexes, we fit the peak  $O_3$  model separately for boys and for girls. Results are shown in Table 4.5.2.1.2. No statistically significant differences were observed.

#### **4.5.2.2 Wheeze**

We defined as cases of wheeze all subjects who reported on annual questionnaire that during the previous 12 months they had wheezed at least 3 days per week for a period of at least one month, or took medication for wheezing, or were seen in a hospital emergency room for wheeze, or had been hospitalized overnight for wheeze. Subjects who reported ever having been diagnosed with asthma were not included in this category.

Potential confounders were screened by the procedure described in Section 2 and the only variable entered into the model was hayfever. Table 4.5.2.2.1 presents the effects of exposure estimated from this model. There are no statistically significant effects. Table 4.5.2.2.2 gives the results of separate analyses for boys and for girls. The logistic regression algorithm failed to converge when community was included in the model, so this variable was not included. No statistically significant differences in risk between boys and girls were found.

#### **4.5.2.3 Bronchitis**

We defined as cases of bronchitis all subjects who reported on the annual questionnaire that during the last 12 months they had a case of bronchitis that lasted more than one week, or that they had more than one occurrence of illness, or that they had taken medication for bronchitis, or that they had been hospitalized for bronchitis. Potential confounders were screened by the procedure described in Section 2, and the variables entered into the model were hayfever, mold or mildew ever in the child's home, and carpet in the child's bedroom. Table 4.5.2.3.1 presents the effects of exposure estimated from this model.  $PM_{10}$  is significantly associated with excess risk. Separate analyses for boys and for girls are reported in Table 4.5.2.3.2. The effect of  $PM_{10}$  is greater in girls than in boys.

#### **4.5.2.4 Cough**

We defined as cases of cough all subjects who reported coughing on annual questionnaire first thing in the morning for at least 3 of the last 12 months. Potential confounders were screened by the procedure described in Section 2, and the variables entered into the model were hayfever, current taking of vitamins, income, and mold or mildew ever in the child's home. Table 4.5.2.4.1 presents the effects of exposure estimated from this model. There are no significant associations with exposure. The estimated relative risk of  $PM_{10}$  exposure is greater in girls than in boys (Table 4.5.2.4.2), but the difference is not statistically significant.

#### **4.5.2.5      Pneumonia**

We defined as cases of pneumonia all subjects who reported having pneumonia during the last 12 months. Potential confounders were screened by the procedure described in Section 2, and the only variable entered into the model was health insurance. There were only 50 pneumonia cases for which we had exposure information. Partly for this reason, the model was badly overfit. Specifically, no community intercepts were statistically significant, nor were race indicators, age, or sex. We dropped these variables from the model, and adjusted only for health insurance. Table 4.5.2.5.1 gives the results. Average  $O_3$  is significantly associated with a decrease in pneumonia. We have no explanation for this finding other than it being a statistical anomaly possibly resulting from multiple tests. Because of the small number of cases, the maximum likelihood algorithm did not converge for boys or girls separately. We therefore fit unadjusted models, containing only the exposure variables as covariates. The results are shown in table 4.5.3.5.2. The boys account for the largest part of the deficit in pneumonia associated with average  $O_3$ .

#### **4.5.2.6      Other Chest Illness**

Subjects were given the opportunity to report the occurrence of chest illnesses which were not asthma, pneumonia, or bronchitis, or for which they did not know the diagnosis; these were specified on the questionnaire as "other chest illness". We defined as cases of other chest illness all subjects who reported that during the last 12 months they had a such an illness that lasted more than one week, or that they had more than one occurrence of illness, or that they had taken medication for such an illness, or that they had been hospitalized for such an illness. Potential confounders were screened by the procedure described in Section 2 and the variables entered into the model were current taking of vitamins, and mold or mildew ever in the child's home. Table 4.5.2.6.1 presents the effects of exposure estimated from this model. There are no significant relationships with pollution exposure. Table 4.5.2.6.2 presents the results of separate fits to boys and to girls. Again there are no significant effects.

#### **4.5.3    Morbidity by Past Individually Constructed Exposure History: Migrants Only**

In many communities, particularly those with high ambient levels of pollution, migrants tend to have less exposure to pollutants than lifelong residents. For this reason the findings of Section 4.5.2 could in principle be explained by confounding of air pollution exposure with migrant status. To address this issue, we fit the models described in Section 4.5.2 to migrants only. Table 4.5.3.1 presents the results. The estimated effects are about the same for migrants as for all subjects taken together for all endpoints.

#### **4.5.4 Morbidity by Past Individually Constructed Exposure History, Adjusted for Physical Activity**

We screened the six physical activity variables described in Section 2.5 for entry into the models described in Section 4.5.2. The only morbidity endpoints for which any of the physical activity variables were significant at the 15% level were cough, and pneumonia. The number of hours spent outdoors during the last two weekends was significant for pneumonia, and the number of hours very physically active during the last two weekends was significant for cough. No interactions between these physical activity variables and exposure were statistically significant. Tables 4.5.4.1 and 4.5.4.2 show the effects of exposure adjusted for physical activity. The results are qualitatively the same as without adjustment.

### **4.6 MORBIDITY ASCERTAINED BY ABSENCE MONITORING**

#### **4.6.1 Descriptive Statistics**

A description of the absence monitoring activity is included in Section 2.3.3. The data included in this analysis represent the period from January 1994 (when complete absence monitoring was established) through December 15, 1994. Most of the absences occurring during that period were recorded. Table 4.6.1.1 presents the number of absences, the number about which interviews were conducted, and the number of interviews in which respiratory illnesses were reported, broken down by sex, race, and grade level. We were able to interview only a modest proportion of the absences reported by the schools. There were two main reasons for this. First, some schools were not well set up to provide these data and thus often failed to provide data in a timely fashion. We had determined from our pilot study that parents were unable to provide reliable information about absences that had occurred more than four weeks before the interview. Therefore, interviews were not attempted for absences reported more than four weeks after their occurrence. Second, to call all the children who were absent turned out to require much more than the 1 FTE budgeted for this activity. So we were not able to call all absences from all schools, but aimed instead for a representative sample. We do believe that the sample called was reasonably representative, so that the proportion of absences we observed to be due to respiratory illness is likely to be close to the true proportion.

Many of the schools provided us with information about the reasons for absences. We did not attempt to interview in cases where we had information that an absence was not illness-related. The results in Table 4.6.1.1 indicate that almost all school absences that resulted in interviews were due to illness, and that about 40% of the illnesses were respiratory in nature. We define an absence to be due to respiratory illness when any of the following symptoms were reported: runny nose, sore throat, cough, wheezing, or asthma. There is no apparent difference in the proportion of illnesses that are respiratory between boys and girls. The proportion is somewhat higher



for blacks, Asians, and American Indians than for other races, but the difference is not statistically significant. The grade-specific results suggest that the proportion of illnesses that are respiratory may decrease slightly with age.

Table 4.6.1.2 presents results by community. The proportion of absences resulting in an interview varies considerably from community to community, reflecting the differences in the timeliness of reports from the various schools. The proportion of illnesses that were respiratory in nature did not vary much from community to community except for Long Beach, where the proportion was much lower. Relatively few interviews were conducted in Long Beach, because reports were less often timely. Thus the apparent reduction in the proportion of absences due to respiratory illness may be due to sampling error.

Table 4.6.1.3 presents absence data by month. As expected, the proportion of illnesses that are respiratory is highest in the winter months. Table 4.6.1.4 presents absence data by day of the week. More absences start on Monday than on any other day. This may be due to the fact that illnesses which start on a weekend and carry over to Monday result in an absence that starts on Monday. It also appears that a smaller proportion of absences that begin on Fridays are due to respiratory illness than those beginning on other days of the week. We have no clear explanation for this finding at this time.

Table 4.6.1.5 presents data by asthmatic or wheezer status. As expected, illnesses suffered by asthmatics are much more likely to be respiratory than illnesses suffered by non-asthmatics. Illnesses suffered by wheezers appear to be equally likely to be respiratory as illnesses suffered by non-wheezers. Both asthmatics and wheezers are more frequently absent than the others.

#### **4.6.2 Risk of Absence Related to Recent Exposure to Air Pollution**

Risks were estimated using a version of the case-crossover design (Maclure, 1991). This is equivalent to a conditional logistic regression in which the days, on which a given subject is eligible to be absent, form a stratum. We fit a total of six models, one for each of  $O_3$ ,  $PM_{10}$  and  $NO_2$  lagged one or two days. Each model also included temperature and humidity lagged one and two days, as well as indicators for the month and the day of the week.

School absences are autocorrelated, that is, if a subject is absent on a given day, the probability of absence the next day is greatly increased. In the analysis presented here, this was compensated for by treating only the first day of each period of absence as failure, and discarding subsequent consecutive absences. Days on which the subject attended school were used as control days.

In standard conditional logistic regression, a subject with  $n$  absences contributes a term whose numerator is the probability that the subject was absent on those  $n$  days, and whose denominator is the sum of the probabilities over all sets of  $n$  days. The number of such sets is too great for the sum to be computed. Following Langholz and Goldstein (1997), we replace this denominator with the sum of the probability for a randomly chosen set of  $n$  control days plus the probability for the days actually absent.

Table 4.6.2.1 presents relative risks of absence due to all causes in relation to peak ambient levels of  $O_3$ ,  $PM_{10}$ , and  $NO_2$  both one and two days prior to the start of the absence.  $NO_2$  lagged two days is significantly associated with increased risk.

Table 4.6.2.2 presents relative risks of absence due to respiratory illness. No statistically significant results are observed. The sample size is much smaller than that for the analysis presented in Table 4.6.2.1, as is reflected in the increased width of the confidence intervals.

Tables 4.6.2.3 and 4.6.2.4 present relative risks of absence due to respiratory illness restricted to wheezers and asthmatics, respectively. No statistically significant results are observed.

#### **4.6.3 Risk of Absence Related to Long-term Exposure to Air Pollution**

Tables 4.6.3.1 and 4.6.3.2 present estimates of the effects of lifetime exposure to peak  $O_3$ ,  $PM_{10}$ ,  $NO_2$ , and average  $O_3$  on the number of absences and the total duration of absences during 1994, respectively. No results are statistically significant. We did not include acid exposure in these analyses because we have no estimates of lifetime acid exposure.

Tables 4.6.3.3 and 4.6.3.4 present analogous results for absences due to respiratory illness. Increased lifetime exposure to  $O_3$  is significantly associated with an increase both in the number of absences due to respiratory illness, and to their total duration.

#### **4.7 PULMONARY FUNCTION AND MORBIDITY IN RELATION TO 1994 AMBIENT LEVELS**

In Section 4.3.1 we presented community level analyses of pulmonary function and morbidity, using as exposures, estimated average ambient levels of pollutants during the interval 1986--1990. Many of these estimates were not as accurate as we would have liked, as they were calculated by interpolating measurements taken at distant sites. During the year 1994, we measured  $O_3$ ,  $PM_{10}$ , and  $NO_2$  on an hourly basis, and several acid species on a two-week integrated basis. These measurements provide a much more accurate picture of the true ambient levels in the 12 communities for 1994 than our

previous estimates did for the time period 1986--1990. For this reason, we repeated our community-level analyses, using the average 1994 levels as the exposures.

Table 4.7.1 presents the results of fitting models including average daily  $O_3$ ,  $PM_{10}$ ,  $NO_2$ , and inorganic acid to 1994 values of  $FEV_1$ . None of the results were statistically significant.

Tables 4.7.2 - 4.7.4 present analogous results for FVC, PEF, and MMEF, respectively. As with  $FEV_1$ , no statistically significant effects were found.

Tables 4.7.5 - 4.7.10 present results for asthma, wheeze, bronchitis, cough, pneumonia, and other chest illness, respectively. No statistically significant results were found, just as with the analyses based on the 1986--1990 estimates.

The significant associations of lifetime exposure to health outcome suggests the importance of either long term exposures or exposures early in life as being etiologically important.

## **4.8 STATUS OF AMBIENT ACIDITY HEALTH EFFECTS ASSESSMENTS**

### **4.8.1 Introduction**

Among the critical tasks set for us by the ARB, was to ascertain the respiratory health consequences of exposure to acidic air pollutants that are present in California atmospheres. When this charge was given by ARB, and accepted by the research team, it was with full recognition by all involved that this was no small challenge. There were a number of questions and technical issues that would have to be addressed during Phase I and the early stages of Phase II before we could begin to meet this goal; we have found that to answer some questions is requiring more time than anticipated and they will not likely be answered until the early part of the longitudinal study (Phase III). These issues and the determinations at which we initially arrived, are briefly discussed below. Also presented within the context of each question, is a brief summary and evaluation of our progress in meeting this goal during Phase II, as well as a summary of our continuing work (Phase III) to address this potentially important public health issue.

It should be noted that the initial decisions were based on extensive reviews of peer-reviewed literature, analysis of the available (albeit limited) ambient air quality data for the predominant acid species present in the study region, and extensive discussions with many outside experts, including the ARB's Scientific Advisory Committee on Acid Deposition and Research Screening Committee, and USC's External Advisory Group for the project. For further information see Phase I Final Report and Addendum (Peters et al., 1992 a, b).

#### **4.8.2 Which Acidic Species Should be the Focus of this Study?**

The first question was which acidic species should be measured based on (a) health effects previously observed in related research, and (b) the pollutants present in California's atmosphere in general, and the South Coast Air Basin in particular.

The dominant form of atmospheric acidity in the study region is vapor phase acid. Nitric acid and hydrochloric acids are the first and second most abundant strong acids, respectively, and formic and acetic acids are the two most abundant weak acids in ambient air. Given that the levels of these air pollutants in the study region are higher than those in other urban areas in the United States, especially in the case of nitric acid, and given that several million people are routinely exposed to these high concentrations it was important to study the effects of these acids.

Unlike other parts of the country, aerosol acidity (especially sulfuric acid) does not contribute substantially to the acidic pollutant burden in Southern California. A possible exception to this might be in areas such as Riverside and Rubidoux where ammonium nitrate (the ammonium ion is acidic) can contribute substantially to fine particles ( $PM_{2.5}$ ). Thus, quantification of ammonium nitrate levels was incorporated into the chemical analysis regimen for  $PM_{2.5}$ .

In the process of answering the first question, it was also concluded that in the apparent absence of health effects from short-term exposure, collection of two-week integrated samples throughout the year would provide accurate seasonal and annual average concentrations of the vapor phase acids, and of  $PM_{2.5}$  from which aerosol acid concentrations could be assayed.

There has been no information in the peer-reviewed literature to suggest any changes to the original decisions regarding which pollutants to study.

#### **4.8.3 What would be the best method for measuring acidic species?**

The second question was related to the methodologies available to measure acidic species. A fundamental concern was that one might not be able to accurately and precisely measure acidic species such as nitric acid due to interference from co-pollutants, and due to limitations in the available technology for measuring these pollutants.

As was discussed in Section 3 of this report, an efficient and reliable instrument was developed and deployed for this project during the first year of Phase II.

No new information has arisen from our work, or the work of others, to suggest that either the measurement methods employed or the two-week integrated measurement approach needs to be adjusted during Phase III.

#### **4.8.4 Can the Effects of Acidic Pollutants be Differentiated From Those of Co-Pollutants?**

The central issue raised by questions three and four was whether an epidemiologic study could be designed such that the effects of acidic pollutants could be differentiated from those of other pollutants. Question three was concerned with whether the accuracy and precision of air quality measurement methods and that of the methods for quantifying health effects were sufficient to be able to identify the effects of ambient acidity. In other words, given that long-term exposure effects of acidic species were likely to be subtle, was the accuracy and precision of methods available to quantify acids and to quantify health endpoints, such as pulmonary function, sufficient to detect subtle effects if they did exist. Question four asked whether there was sufficient spatial and temporal differences in the relative concentrations of atmospheric acidity and other pollutants, particularly ozone and/or PM<sub>10</sub>, such that the effects of acidic species alone could be identified and quantified.

At the end of Phase I, the answer to question three was affirmative. Now, at the close of Phase II, we are confident that we have achieved the requisite level of accuracy and precision for acidic measurements, and for pulmonary function tests. What we have not been able to adequately evaluate in Phase II is whether, even with high quality measurements, subtle effects of acids will be detectable. In part, this is due to the paucity of reliable historical ambient acidity data. The lack of historical data precluded generating lifetime acid exposure estimates for individuals (discussed in Section 3.2.1), and the community level exposure estimates for acids had to rely on the one year of concurrent acid measurements that we collected during 1994. Phase II pulmonary function measurements were made in the Spring of 1993 and 1994. Analysis of the relationship between pulmonary function and long-term exposures to any of the pollutants, requires that current pulmonary function be evaluated in terms of past exposure, even if that exposure is the immediate past year. Obviously, by the end of Phase II, we did not have the data to conduct such analyses for acids. These analyses will be performed in the early part of Phase III, using 1995 pulmonary function data and the 1994 acid data. In addition, the absence monitoring data and questionnaire data will be analyzed using the collected acid measurements.

The issue that question four addresses is related to the fact that the levels of pollutants such as ozone and nitric acid are often highly correlated both temporally and spatially. This high degree of correlation would likely preclude the ability to tease apart the health effects of one pollutant from another. Thus, to determine the effects of an individual pollutant, it was essential that we identify communities with extremes of exposure for the different pollutants, and that the communities together represented a matrix of exposures that optimally included at least one community with high levels of one pollutant and relatively low levels of the other pollutants. The extent of spatial

correlations are more important than temporal correlation for distinguishing communities. The community selection process is detailed in Phase I Final Report and the Addendum to that Report (Peters et al., 1992 a, b).

During Phase I, review of the available air quality data indicated that, as expected, a strong correlation existed for nitric acid and ozone at most of the sites for which there were data. However, the relationship between the species varied between sites (the slope of the regression line was quite different at certain low acid sites such as Rubidoux). Each of the twelve communities finally selected represented a potentially important combination of high or low ambient concentrations of the four pollutants (Ozone, NO<sub>2</sub>, PM<sub>10</sub>, and strong vapor phase acids (nitric and hydrochloric acids combined)). At the end of Phase I, we concluded that no direct and distinct assessment of the health importance of ambient acidic exposures alone could be made because there were no identified locations in Southern California characterized by significant ambient vapor phase strong acid levels in the absence of ozone or PM<sub>10</sub> ( a so-called "low-low-high" community). However, we believed that because of the unique situation existing at Rubidoux and Riverside, indirect comparisons of the relative contribution to health effects of ambient acidity could be made from the communities selected for study. The idea was that comparisons of the response of school children in communities exhibiting similar levels of ozone and PM<sub>10</sub> and differing levels of acid vapor would elucidate any significant acid effects.

The data from Phase II, presented and discussed in Sections 3.2.3 and 3.2.4, indicate that there were notable differences in the patterns of acid levels among the 12 communities. These deviations from expected, especially the finding of higher levels of acids at Riverside and Mira Loma than expected, may compromise the study's ability to separate the health effects of inorganic acids from those caused by the other pollutants. During Phase III we will further evaluate the effects of acids primarily by assessing how acidic levels influence lung growth rate as measured by our annual assessment of pulmonary function.

The overall growth rate for 4 years of follow-up can be assessed using the acid measurements for the 4 year period. In addition, each of the annual growth increments (4) can be evaluated against the annual acid exposure. These approaches should provide the most objective and sensitive assessments of whether acid exposures are associated with slowed lung growth. The questionnaire assessment of symptoms and conditions, and the absence monitoring data on respiratory illnesses will provide important supplementary data. While the correlation between O<sub>3</sub> and acid vapor is quite high, we believe that if acid vapor is causing significant adverse health effects, it will be possible to differentiate them from other exposures.

These analyses should be completed by the end of 1997.

## 4.9 DISCUSSION

### 4.9.1 Introduction

Overall disease prevalence or pulmonary function values did not differ between communities with different levels or profiles of pollution, ruling out profound respiratory effects of air pollution. There were subtle, statistically significant relationships between certain pollutants and health effects measured by pulmonary function, questionnaire-based respiratory disease prevalence and absence-based morbidity monitoring when the data were analyzed at the individual level.

The exposure assessments on which we place the most reliance are on the lifetime exposure history based on individual residential histories and on the measurements made at the 12 monitoring stations during 1993-1994. On a theoretical level, we therefore believe that any associations seen between these exposures and health outcomes would be the most credible. We believe our estimates of lifetime ozone exposure are more accurate than either  $PM_{10}$  or  $NO_2$  because they are based on longer and more extensive sampling data. If an exposure to an air pollutant is associated with an effect on pulmonary function, the more accurately the exposure is estimated, the more likely an association will be seen. This could be the explanation for the clearer relationships between  $O_3$  exposure and changes in lung function. It is also important to consider the three types of health outcomes with respect to predicted effects deriving from the study design.

Pulmonary Function. In our study, our measurements of pulmonary function are the most objective assessment of the current status of the structure and function of the lung. We made these measurements to assess the cumulative effects of any insults the lung had been subjected to in the life of the child. These measurements were made at a time to minimize any acute effects of air pollution (during the Spring months when pollution levels are low and in the morning when pollution levels are at their lowest). We therefore believe that the most relevant exposure assessment for pulmonary function is the lifetime exposure. Exposures early in life could be more important than recent exposures. The fact that lifetime exposure was more closely associated with pulmonary function effects than recent exposure suggests that either early exposures or cumulative exposures are the most important.

Questionnaire-based Respiratory Symptoms and Conditions. Our working assumption is that the asthmatic state is not caused by air pollution but that the frequency and severity of asthmatic episodes could be related to air pollution episodes. For wheeze, cough, bronchitis, and pneumonia it is reasonable to assume that recent exposures could be important. Likewise, it is reasonable to assume that lifetime exposure could predispose one to be more sensitive to recent exposures. It is therefore, difficult to select specific temporal exposures as the most logical exposure parameter for assessing questionnaire based symptoms.

School Absence-based Morbidity. The same arguments made for questionnaire-based symptoms or conditions apply to absence monitoring. Cumulative effects of pollution could result in a lung that is more sensitive to the effects of recent pollution, or recent pollution could directly cause morbidity.

#### 4.9.2 Respiratory Morbidity

We found little association between outdoor air pollution exposure and prevalence of asthma, bronchitis or pneumonia whether we defined exposure over the subject's entire lifetime, over five fairly recent years, or by one year's very recent air quality data collected specifically to relate with our health data. We found only one statistically significant relationship, between risk of bronchitis and lifetime estimated  $PM_{10}$  exposure. This might be attributed to chance, given that a large number of associations were examined. When multiple statistical tests are performed, statistically significant outcomes occur by chance alone. On the other hand, the association of children's bronchitis with particulate pollution was one of relatively few specific health/pollution relationships found to be significant in the Harvard Six- City Study, when analyzed across cities (though not when analyzed across higher- and lower-pollution areas within cities) [Ware et al., 1986; Dockery et al., 1989]. Given this consistency between ours and earlier findings in children, as well as the numerous reports of acute and chronic health effects of particulate pollution in adults [Bascom et al., 1996], the possibility of a casual relationship between bronchitis and some component of  $PM_{10}$  must be seriously considered.

The overall results concerning outdoor air pollution are somewhat reassuring, in that we found no consistent or large excesses of morbidity in subjects who lived in the most polluted communities and/or had the highest estimated lifetime exposures. This might indicate (a) little effect of even the most severe outdoor pollution, (b) an increase of other risk factors in cleaner communities, offsetting any reduced risk from pollution, or (c) our inability to detect important effects, because of exposure misclassification and/or inadequate sensitivity of health measures. Other possible risk factors notable in the cleanest communities included relatively cool summers in Lompoc and Santa Maria, and relatively cold winters in Atascadero, as well as farmlands and grasslands, increasing the potential for airborne allergen or pesticide exposure, near all three communities. However, each of those potential risks was present in one or more polluted communities also. Thus, a "tradeoff" of effects between other risk factors and air pollution seems unlikely, although it cannot be ruled out. We sought to minimize exposure misclassification through an intensive outdoor air monitoring effort. However, individuals' exposures indoors (where typical California children spend 85% or more of their time [Wiley, 1991]) may not have reflected outdoor conditions, and individuals' outdoor exposures should have varied with their time-activity patterns, which we could document only crudely. Thus, we cannot rule out the possibility that either indoor



exposures or outdoor activity varied systematically with outdoor air quality. If so, we might have misclassified many individuals' exposures, and thereby failed to detect real effects of outdoor pollution.

#### 4.9.3 Pulmonary Function

**Community Comparisons.** The average lung function values, as a percent of predicted, adjusted for measurable influences (such as age, size and gender) operating at the individual level other than pollution exposure, did not differ significantly between communities with different levels or profiles of air pollution. That is, communities with the highest exposures did not show the lowest percent of predicted. That finding argues against profound long-term respiratory effects of any of the pollutants studied, at levels experienced in Southern California in recent years (As previously mentioned, Southern California levels are among the highest in the U.S.). Nevertheless, the results when analyzed by individualized exposure histories, showed subtle statistically significant relationships between estimated lifetime exposures and lung function losses which may have public health significance.

**Likelihood of a Cumulative Effect from Ozone.** The exposure assessments which we consider most reliable are the lifetime exposure histories based on residential histories, and the measurements made at the 12 community monitoring stations during 1993-94. We consider the lifetime exposure estimates to be more accurate for O<sub>3</sub> than for PM<sub>10</sub> or NO<sub>2</sub>. On a theoretical level, therefore, we consider any associations between lifetime or recent O<sub>3</sub> exposure estimates and health outcomes to be most credible. On this basis, both the significant negative effect of lifetime O<sub>3</sub> exposure and the lack of measurable effect from recent O<sub>3</sub> exposure are credible findings. Taken together, they argue that lung dysfunction due to O<sub>3</sub> either is cumulative over a child's lifetime, or is most pronounced in early years. The tendency toward increasingly negative regression coefficients with increasing age argues that the unfavorable effect of O<sub>3</sub> exposure is cumulative, continuing throughout childhood. This is the primary hypothesis for our continuing study of this cohort. The significant results for all flow-related lung function variables (including FEV<sub>1</sub>), in contrast with the completely non-significant results for FVC, suggest that the cumulative O<sub>3</sub> effect is primarily obstructive rather than restrictive.

Regression results for boys of all ages suggest that the magnitude of the effect is not trivial. Boys' estimated effect of lifetime average peak O<sub>3</sub> exposure was -3.22 ml/ppb (95% CI -5.47, -0.96). The relevant 5th and 95th percentile exposure concentration estimates were 41 ppb and 104 ppb respectively. Thus, a typical FEV<sub>1</sub> loss associated with the high end of the exposure range throughout all 12 communities, as compared with the low end, may be estimated as  $(104 - 41) \times 3.22 = 203$  ml. This represents approximately 5-10% of the FEV<sub>1</sub>. If a loss of this size persisted into adulthood, and no compensatory slowing of function loss with aging occurred, the excess risk of disabling chronic disease and shortened lifespan might be appreciable.

[Sorlie et al., 1989]. The consistently lesser effects in girls than in boys are plausibly explained by girls' lower levels of physical activity and greater proportion of time spent indoors, both of which would tend to reduce their effective doses of  $O_3$ . The significant sex differences, as well as the larger regression slopes in analyses which take individual activity levels into account, support the possibility of a cumulative effect of  $O_3$  on lung function. The influence of time-activity patterns on effective pollutant doses and health outcomes are being investigated in more detail in the longitudinal study (Phase III).

**Other Possible Explanations for Regression Results.** Given a real association between an exposure and lung dysfunction, the more accurate the exposure estimate, the better the chance of detecting the association statistically. Thus, our finding of an  $O_3$  effect but not a  $PM_{10}$  effect on lung function (in contrast to our findings of  $PM_{10}$  effects in health questionnaire data and numerous prior findings of particulate effects in regions other than Southern California) might be explained by better exposure assessment for  $O_3$ , and not by greater toxicity of  $O_3$ . On the other hand, extensive toxicological evidence argues for a greater effect of  $O_3$ , as discussed later. Regardless of exposure assessment accuracy, the positive association of  $NO_2$  exposure with lung function is biologically implausible, and therefore probably spurious. The complex chemical interactions in the atmosphere between  $NO_2$  and  $O_3$  may be partially involved in this paradox.

In principle, our experimental design could not distinguish acute reversible pulmonary function effects of recent air pollution exposure from the chronic effects of interest. As mentioned previously, we minimized the possibility of acute effects by scheduling health testing to avoid high- $O_3$  hours or seasons. A concurrent study evaluated elementary school children's acute effects in three communities with contrasting air quality, whose air quality resembled three of our communities. It showed a small significant negative association between morning FVC measurements and the past 24 hours' level of particulate pollution or  $NO_2$ , but no significant association of  $O_3$  with FVC, and no significant association of any pollutant with  $FEV_1$  [Linn et al., 1996]. Based on those results, our  $O_3$ -related lung function decrements were not likely caused by acute effects.

The counterintuitive finding of significantly increased  $FEV_1$  in active smokers requires explanation, because it reflects on the credibility of chronic air pollution effects detected by similar means. A likely explanation is bias due to residual confounding of active smoking with height, weight, and age. Active smokers tend to be taller, heavier, and older than nonsmokers in our population. The linear terms for height, weight, and age in our analytical models may not completely capture the effect of these variables on  $FEV_1$ . Although quadratic, logarithmic, or other transformations might improve control of residual confounding somewhat, the precise relationships of height, weight, and age to  $FEV_1$  are undoubtedly too complex to be modeled exactly. Because these three variables are powerful predictors of  $FEV_1$ , even a small degree of such residual confounding can produce a noticeable bias. Similar biases in the estimation of air

pollution effects are unlikely, because systematic relationships between pollution exposure and height, weight, or age are unlikely.

Notwithstanding the likelihood of bias in the analysis, at least part of the nonsmokers' function "deficit" may be real, assuming that subjects with initially poorer lung function were less likely to take up smoking, and that most of those who did smoke had not done so long enough to cause appreciable function loss. Previous evidence concerning effects of active smoking by young people is mixed, but shows some consistency with those assumptions. Gold et al., [1996], examining 10- to 18-year-old subjects in the Harvard Six-City Study, found higher FVC in smokers compared to nonsmokers, and similar or higher FEV<sub>1</sub>, but lower MMEF and FEV<sub>1</sub>/FVC ratios. Tager et al., [1985] found generally lower FEV<sub>1</sub> in smoking children and adolescents, but little difference between nonsmokers and smokers with low total consumption. Beck et al., [1981], comparing young smokers and nonsmokers, found higher FEV<sub>1</sub> in female smokers aged 7-14 and lower FEV<sub>1</sub> in their male counterparts, but the gender differences reversed in the 15-24 age range.

**Comparison with Previous Findings: Toxicology.** Evidence from acute and chronic animal exposure studies and acute studies of human volunteers leaves little doubt that O<sub>3</sub> can cause unfavorable respiratory effects at exposure levels within or slightly above the ambient range. This evidence has been reviewed recently [Bates, 1995a, 1995b; Bascom et al., 1996]. Multiple animal studies have shown apparently irreversible lung pathology, usually most evident in small peripheral airways, after several weeks to several months of intermittent exposures simulating diurnal variations of ambient O<sub>3</sub>, with maximum concentrations of 200 to 250 ppb. Heavily exercising adult humans exposed in laboratories for periods of 6 hr or longer to O<sub>3</sub> concentrations as low as 80-120 ppb have shown acute lung dysfunction [Horstman et al., 1990; Linn et al., 1994] and lung inflammation [Devlin et al., 1991]. Dose-response relationships have been worked out in detail for lung dysfunction [McDonnell et al., 1983], and exposures to O<sub>3</sub>-containing ambient pollution have been shown to evoke similar responses [Avol et al., 1984; Specter et al., 1991]. The acute lung dysfunction is primarily restrictive rather than obstructive, in contrast to the apparent long-term effects in our subjects. However, inflammatory responses are likely more important than acute lung dysfunction as precursors of chronic effects; and prolonged lung inflammation may lead to either restrictive or obstructive changes [Bascom et al., 1996].

In contrast to O<sub>3</sub>, NO<sub>2</sub> has shown no effects at ambient-like concentrations in some animal and human exposure studies, and relatively mild and subtle effects in others [Bascom et al., 1996, part II]. Some investigators have reported small acute lung function losses and/or increased bronchial reactivity in asthmatic volunteers exposed briefly to 500 ppb or less [Bylin et al., 1985; Moshenin et al., 1987], but the most extensive human exposure studies have not found such effects to be statistically significant, although in some instances a minority of subjects appeared to be reactive [Linn et al., 1985; Roger et al., 1990]. In any event, toxicologic evidence is consistent

with our present findings, showing  $\text{NO}_2$  to be generally far less toxic than  $\text{O}_3$  at ambient concentrations.

Toxicologic assessment of  $\text{PM}_{10}$  is considerably more complicated. The composition of  $\text{PM}_{10}$  exhibits considerable geographic variation, even within Southern California, and actual ambient  $\text{PM}_{10}$  pollution is far more complex physically and chemically than particulate matter generated artificially for exposure studies. Many controlled exposure studies have employed sulfuric acid aerosol, which is suspected to be the most toxic component of  $\text{PM}_{10}$  in many locations but is not important in Southern California. Human studies have shown no more than small or equivocal respiratory effects of sulfuric acid unless concentrations far exceed the ambient range [Horstman et al., 1982; Utell et al., 1984; Spektor et al., 1985; Aris et al., 1990; Frampton et al., 1992]. Prolonged, repeated exposures to combined sulfuric acid and  $\text{O}_3$  showed only marginally increased pulmonary function effects, as compared with  $\text{O}_3$  alone [Linn et al., 1994]. This finding might be relevant to the parts of Southern California where a different strong acid - nitric acid vapor - may accompany  $\text{O}_3$ . In general, the available toxicologic evidence concerning  $\text{PM}_{10}$  components is consistent with our finding of less effect on pulmonary function from  $\text{PM}_{10}$  than from  $\text{O}_3$ . However, the generally negative evidence is less convincing for  $\text{PM}_{10}$  than for  $\text{NO}_2$ , because of the possibility that toxicologic studies may have overlooked some highly toxic  $\text{PM}_{10}$  components, and because of the extensive epidemiologic evidence suggesting unfavorable effects of  $\text{PM}_{10}$  (see below).

**Comparison with Previous Findings: Epidemiology.** The only previous large-scale comparison of lung function across different parts of Southern California, suggested lower pulmonary function levels and more rapid loss rates in adults living in more polluted communities [Detels et al., 1987]. The most polluted study sites were high in  $\text{PM}_{10}$  as well as  $\text{O}_3$ , so the observed effects might be attributed to either. Although not directly comparable because of subjects' age differences, these findings appear consistent with our finding of an unfavorable long-term effect of  $\text{O}_3$ . Further support is provided by an analysis of data collected nationwide in the NHANES II survey, which suggested decreases in adults' lung function with increasing annual average  $\text{O}_3$  concentration in their areas of residence [Schwartz, 1989]. A preliminary evaluation of lung function in U.S. Military Academy cadets in relation to long-term  $\text{O}_3$  exposure [Ito et al., 1993] suggested FEV<sub>1</sub> loss of 2.9 ml/ppb. Although the relationship was of marginal statistical significance, the regression estimate is strikingly close to our estimate of 3.2 ml/ppb for boys. Thus, prior epidemiologic evidence concerning long-term  $\text{O}_3$  effects, albeit very limited, is in good agreement with our findings.

We are not aware of prior epidemiologic studies which assessed effects of long-term  $\text{NO}_2$  exposure on lung function. Multiple studies of symptom and illness rates in relation to indoor or outdoor  $\text{NO}_2$  pollution have yielded mixed results [Bascom et al., 1996, part II].

The most directly relevant prior epidemiologic studies have been conducted away from Southern California, and have been concerned primarily with particulate pollution. The Harvard-Six City Study showed no significant association between recent or lifetime particulate exposure and FVC or FEV<sub>1</sub> [Ware et al., 1986; Dockery et al., 1989], although it did show an association between questionnaire-reported bronchitis and particulate exposure, consistent with our findings. All six cities were low in O<sub>3</sub>, in comparison with our most polluted communities. In the subsequent 24 City Study, preliminary results showed that bronchitic symptoms were again associated with particulate exposure (specifically, with the strong acid component) [Dockery et al., 1993]; and lung function was also reduced in association with those exposures [Raizenne et al., 1993], in contrast with the Harvard-Six City Study. The predicted loss due to lifetime residence in the community with highest strong acid particulate concentration, relative to the cleanest community, was about 3% for FVC and FEV<sub>1</sub>. Ozone effects were not reported. Most of the 24 cities had relatively little O<sub>3</sub>, compared to our most polluted Southern California communities. In a study of preadolescent children in 10 rural Canadian communities, five of which had elevated O<sub>3</sub> and particulate sulfate, significant FVC and FEV<sub>1</sub> decrements between 1% and 2% were found in the five polluted communities [Stern et al., 1994]. No significant excess respiratory morbidity was reported on questionnaires from the five polluted communities. Their annual mean of 1-hr daily O<sub>3</sub> peaks was 46 ppb, less than half the value in our most polluted communities. Comparison of strong acid or particulate concentrations between studies is difficult because of differences in the atmospheres and in the measurement techniques.

Each of the above most directly relevant studies shows some consistency and some inconsistency with our study. The estimated FEV<sub>1</sub> loss from living in the most polluted community was roughly the same in our study and the 24 City Study, and about half as large in the Canadian study, where maximum pollution levels were probably lower. Thus, the absence of significant lung function loss in the Harvard Six-City Study seems anomalous. Possibly the later three studies attained greater precision in pulmonary function measurements, by taking advantage of experience from the Harvard Six-City Study as well as subsequent technological improvements. Failure to find a bronchitis/particulate pollution relationship is an apparent anomaly in the Canadian study, perhaps explainable by a lower range of exposures. Our finding of an effect on FEV<sub>1</sub> (and flow rates) but not FVC contrasts with the Canadian and 24 City studies. Such an outcome could be expected if O<sub>3</sub> (the dominant exposure in our population) had a predominantly obstructive effect, but acid sulfate (the dominant exposure in the other populations) had a predominantly restrictive or mixed effect, and particulate matter without acid sulfate (as experienced by our population) had little effect even at high concentrations. Other toxicologic and epidemiologic evidence seems inadequate to support or refute this hypothesis. Another possibility is that all four populations experienced long-term respiratory morbidity and lung dysfunction from higher-level exposures, regardless which type of pollution predominated in their

localities, and that different results from different studies were mostly the result of chance, since all the health effects were small and all the measurements were being pushed to their limits of sensitivity.

#### **4.9.4 Discussion of Absence Monitoring**

Our first year of absence monitoring yielded a few statistically significant results most notably related to lifetime ozone exposure. Variation in air pollution levels on a short term basis (one or two days) was not related to the frequency of respiratory illnesses. This may be because air pollution levels were not high enough to cause respiratory illnesses or it may be that acute responses (if present) were not severe enough to cause a student to stay home from school. On the other hand, lifetime ozone exposure was associated with frequency and severity of respiratory illness. This finding, if borne out in Phase III, suggests that chronic exposure or even exposure early in life is the important factor.

Since we believe that the most likely effect of air pollution on absence operates through respiratory illness and that absence rates due to other causes are virtually unaffected, it follows that the power of an analysis based on the total number of absences is substantially reduced by the fact that about 60% of the absences are of a sort whose frequency is probably unaffected by exposure. By restricting the analysis to absences due to respiratory illness, power is enhanced. Our sampling scheme for respiratory illness resulted in only a small proportion of those illnesses being ascertained, which reduced the power of analyses based on these data.

The procedure by which respiratory illnesses were ascertained was unavoidably somewhat non-random. This may have introduced some artifacts into the results of the analyses based on respiratory illnesses.

In Phase III, as the volume of absence data grows, the power of analyses based on all absences will increase. As we increase our effort to sample respiratory illnesses, the power and accuracy of analyses based on respiratory illness will increase as well.

#### **4.10 Conclusions on Health Effects**

On the basis of our Phase II results we conclude that long-term exposure to  $O_3$  is associated with subtle adverse changes in pulmonary function and higher rates and duration of respiratory illnesses as assessed by school absence monitoring. Long-term exposure to  $PM_{10}$  is associated with a greater prevalence of bronchitis as assessed by questionnaire.

The Phase III follow-up of these children is underway to clarify the causality of these relationships and to determine permanency. Our Phase II findings have provided several important issues to be pursued. For example, we found that boys spend more

time outdoors, and more time engaged in strenuous physical activity, than girls do. The difference in the magnitude of the ozone effect between boys and girls, and its potential relationship with differences in time activity patterns will be addressed more precisely with models that adjust exposure measurements by taking time outdoors and physical activity into account.

The findings that adverse effects of exposure tend to be greater in older children is suggestive of a cumulative effect. Longitudinal methods will enable us to estimate the magnitude of this effect as a function of time. This will provide us with a description of changes in lung growth which may occur in response to air pollution exposure.

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**TABLE 4.1.1.1 NUMBERS OF SUBJECTS AND TYPES OF INFORMATION AVAILABLE  
BY COMMUNITY**

Community	Number of Subjects with Questionnaire Information	Number of Subjects with Pulmonary Function Information	Number of Subjects with Physical Activity Information	Number of Subjects with more than 50% Complete Ozone Exposure History
Alpine	301	274	281	231
Lake Elsinore	317	283	290	238
Lake Arrowhead	344	301	304	267
Lancaster	271	243	233	206
Lompoc	311	253	247	212
Long Beach	326	283	282	243
Mira Loma	312	287	292	236
Riverside	369	320	317	277
San Dimas	302	267	256	232
Atascadero	261	230	220	212
Santa Maria	311	248	231	230
Upland	276	259	237	226
<b>TOTAL</b>	<b>3701</b>	<b>3248</b>	<b>3190</b>	<b>2810</b>

**TABLE 4.1.1.2 DISTRIBUTION OF AGE BY COMMUNITY (%)**

Community	AGE (YEARS)									
	8	9	10	11	12	13	14	15	16	17
Alpine	0.0	28.2	22.0	1.1	13.6	11.4	0.0	12.1	11.7	0.0
Lake Elsinore	0.3	18.7	25.2	1.7	9.3	16.2	1.0	14.1	11.7	1.7
Lake Arrowhead	0.0	20.3	26.3	2.7	12.3	13.7	0.3	11.7	11.0	1.0
Lancaster	0.0	20.6	29.2	2.9	11.1	12.4	1.2	12.4	10.3	0.0
Lompoc	0.0	13.1	27.8	1.6	12.3	13.9	0.8	14.3	14.3	1.6
Long Beach	0.0	25.3	27.4	2.8	12.3	9.8	0.4	13.3	8.1	0.4
Mira Loma	0.0	29.1	20.9	3.1	14.7	10.3	0.7	13.4	6.9	0.0
Riverside	0.3	24.7	23.4	0.7	15.9	10.0	0.3	12.8	10.9	0.9
San Dimas	0.0	22.0	24.2	1.8	14.3	11.7	0.7	9.2	13.9	2.2
Atascadero	0.0	14.4	33.5	0.9	9.1	17.0	1.7	9.1	14.4	0.0
Santa Maria	0.4	17.7	26.7	2.8	9.4	12.9	2.4	15.3	12.2	0.4
Upland	0.4	26.9	20.7	1.5	13.4	8.8	0.4	11.5	13.0	1.9

**TABLE 4.1.1.3 DISTRIBUTION OF ETHNICITY AND SEX BY COMMUNITY (%)**

Community	RACE						Percent Hispanic	SEX	
	White	Black	Asian	American Indian	Other	Mixed		Female	Male
Alpine	83.9	0.4	0.7	2.6	6.4	6.0	12.9	50.2	49.8
Lake Elsinore	76.3	2.7	1.7	1.7	11.7	6.0	24.1	46.4	53.6
Lake Arrowhead	83.7	0.6	1.5	0.9	7.1	6.2	16.3	51.5	48.5
Lancaster	70.9	6.1	2.5	0.8	15.2	4.5	26.8	54.4	45.6
Lompoc	72.6	8.6	0.8	1.5	12.4	4.1	18.9	49.5	50.5
Long Beach	37.7	15.8	22.2	0.3	14.4	9.4	22.7	52.1	47.9
Mira Loma	66.9	1.1	1.1	0.7	23.9	6.3	34.3	53.6	46.4
Riverside	43.5	14.1	6.2	2.9	26.5	6.8	39.0	53.1	46.9
San Dimas	62.3	5.5	8.7	1.0	18.3	4.2	29.2	52.4	47.6
Atascadero	83.9	0.4	0.4	0.8	4.4	10.0	11.5	58.8	41.2
Santa Maria	46.4	1.6	2.8	1.2	41.7	6.3	59.6	51.4	48.6
Upland	69.3	2.7	8.8	0.4	11.5	7.3	16.6	50.6	49.4

TABLE 4.1.1.4 DISTRIBUTION OF ANNUAL FAMILY INCOME BY COMMUNITY (%)

	Annual Income						
	< 7,700	7,500 - 14,999	15,000 - 29,999	30,000 - 49,999	50,000 - 99,999	> 100,000	Don't Know
Alpine	4.2	7.6	11.7	25.0	33.7	9.1	8.7
Lake Elsinore	4.8	10.7	16.2	27.5	26.1	1.7	13.1
Lake Arrowhead	4.7	8.7	12.8	28.4	31.2	7.8	6.5
Lancaster	8.0	15.1	10.9	24.3	29.7	3.4	8.8
Lompoc	0.7	3.9	12.5	36.8	35.0	1.1	10.0
Long Beach	6.6	12.5	10.0	23.2	25.3	10.0	12.5
Mira Loma	5.6	9.5	18.7	21.8	27.5	4.6	12.3
Riverside	8.5	10.4	20.1	24.2	20.4	4.4	12.0
San Dimas	4.9	5.7	16.2	24.5	32.1	6.8	10.2
Atascadero	3.5	7.8	14.2	27.6	38.4	2.6	6.0
Santa Maria	11.7	16.9	24.8	23.7	13.9	0.8	8.3
Upland	0.8	2.4	5.5	12.3	53.0	18.6	7.5

**TABLE 4.1.1.5 DISTRIBUTION OF MIGRANT STATUS BY COMMUNITY (%)**

Community	Among Migrants		
	Percent Migrant	Percent Born in U.S. Outside California	Percent Born Outside U.S.
Alpine	74.7	17.6	4.1
Lake Elsinore	81.3	17.6	4.5
Lake Arrowhead	82.4	15.9	4.1
Lancaster	74.5	27.5	8.6
Lompoc	75.3	51.1	6.5
Long Beach	54.7	24.8	27.3
Mira Loma	77.6	8.6	6.3
Riverside	65.1	27.0	18.5
San Dimas	70.1	17.1	9.3
Atascadero	65.6	15.2	1.8
Santa Maria	55.3	19.3	30.7
Upland	71.4	19.5	6.0



**TABLE 4.1.2.1 DISTRIBUTION OF TYPE OF RESIDENCE BY COMMUNITY (%)**

Community	TYPE OF RESIDENCE					
	Single Family Home	Building for 2 - 4 Families	Building for 5 - 10 Families	Building for More Than 10 Families	Mobil Home or Trailer	Other
Alpine	81.6	6.1	2.9	4.3	3.6	1.4
Lake Elsinore	79.9	3.8	2.4	0.3	12.0	1.7
Lake Arrowhead	95.3	2.5	0.9	0.0	0.9	0.3
Lancaster	85.0	4.6	4.2	2.1	2.5	1.7
Lompoc	90.2	2.6	2.3	0.4	3.0	1.1
Long Beach	73.9	13.2	5.5	4.4	0.4	2.2
Mira Loma	89.7	1.7	1.0	0.0	6.5	1.0
Riverside	79.6	7.6	4.0	4.6	0.9	3.3
San Dimas	82.2	5.3	4.6	2.5	2.1	3.2
Atascadero	89.2	1.7	2.1	0.4	4.6	2.1
Santa Maria	71.9	13.1	4.2	5.0	2.3	3.1
Upland	92.8	5.6	1.2	0.4	0.0	0.0

TABLE 4.1.2.2 DISTRIBUTION OF DATE OF RESIDENCE CONSTRUCTION BY COMMUNITY (%)

Community	DATE OF RESIDENCE CONSTRUCTION							Don't Know
	1990's	1980's	1970's	1960's	1950's	1940's	Before 1940	
Alpine	14.9	33.0	23.3	10.8	5.9	3.1	2.4	6.6
Lake Elsinore	16.3	55.1	12.4	5.5	2.3	0.7	0.7	7.2
Lake Arrowhead	10.8	22.8	25.5	13.5	6.3	5.1	6.9	9.3
Lancaster	13.3	38.7	16.1	6.5	14.5	2.4	0.4	8.1
Lompoc	4.2	19.2	12.6	35.0	11.2	1.4	1.8	14.7
Long Beach	2.6	5.9	6.2	5.2	14.0	23.4	17.2	25.7
Mira Loma	3.4	38.1	22.2	10.8	8.1	1.7	0.7	15.2
Riverside	6.7	20.5	14.2	17.3	11.0	4.1	2.9	23.4
San Dimas	3.8	19.0	27.3	21.8	8.3	2.8	5.5	11.4
Atascadero	9.3	38.7	25.8	8.1	4.0	3.2	5.2	5.7
Santa Maria	7.6	19.8	13.7	12.6	10.4	5.4	5.8	24.8
Upland	5.6	44.6	27.7	11.2	4.9	0.4	1.1	4.5

**TABLE 4.1.2.3 DISTRIBUTION OF NUMBER OF BEDROOMS IN RESIDENCE BY COMMUNITY (%)**

Community	NUMBER OF BEDROOMS IN RESIDENCE					
	0	1	2	3	4	5
Alpine	0.0	2.1	18.1	45.8	28.8	4.2
Lake Elsinore	0.6	1.6	11.6	53.7	27.3	4.8
Lake Arrowhead	0.0	2.7	12.8	46.5	26.9	8.6
Lancaster	0.0	0.0	16.1	40.4	38.4	4.3
Lompoc	0.0	0.3	7.2	48.8	38.6	4.8
Long Beach	0.3	5.4	28.8	41.5	19.4	4.4
Mira Loma	0.7	2.3	8.3	45.4	35.8	6.6
Riverside	0.3	2.8	23.2	39.0	29.1	4.2
San Dimas	0.0	0.4	13.8	46.4	32.9	5.2
Atascadero	0.0	0.0	11.8	58.8	23.5	5.5
Santa Maria	0.7	3.2	24.2	51.9	16.8	3.2
Upland	0.0	0.4	5.9	22.6	58.9	10.0

**TABLE 4.1.2.4 MISCELLANEOUS RESIDENTIAL DATA BY COMMUNITY (%)**

Community	One or More Pets	Five or More House Plants	Gas Stove	Microwave	Air Conditioning	Carpet in Child's Bedroom
Alpine	86.1	33.3	46.1	94.9	74.0	92.1
Lake Elsinore	85.6	42.3	76.1	94.2	90.5	89.2
Lake Arrowhead	84.4	44.4	86.0	92.1	8.8	93.4
Lancaster	71.2	46.9	88.3	95.7	94.9	91.9
Lompoc	73.1	46.9	82.3	94.0	2.7	83.7
Long Beach	56.8	38.2	83.8	87.6	33.4	77.8
Mira Loma	87.9	30.9	92.5	91.2	83.6	86.5
Riverside	72.2	32.8	85.9	87.1	82.5	87.4
San Dimas	72.0	28.8	88.3	93.5	86.2	91.1
Atascadero	91.2	49.0	77.1	94.5	66.0	91.8
Santa Maria	52.4	38.2	81.6	79.9	7.5	81.4
Upland	76.8	43.0	71.7	98.2	96.3	93.3

TABLE 4.1.2.5 DISTRIBUTION OF METHODS OF LIGHTING GAS STOVE BY COMMUNITY (%)

Community	METHODS OF LIGHTING GAS STOVE			
	Matches	Pilot Light	Electric Ignition	Other
Alpine	5.3	34.4	58.8	1.5
Lake Elsinore	2.2	35.7	61.3	0.9
Lake Arrowhead	5.2	37.7	56.8	0.4
Lancaster	2.3	31.7	65.6	0.5
Lompoc	2.1	33.5	64.5	0.0
Long Beach	7.2	46.8	45.6	0.4
Mira Loma	3.6	35.4	59.9	1.1
Riverside	4.4	51.0	44.0	0.7
San Dimas	3.2	41.8	55.0	0.0
Atascadero	0.5	31.9	66.5	1.1
Santa Maria	6.0	44.4	48.7	0.9
Upland	2.6	24.9	72.0	0.5

**TABLE 4.2.1 MEAN LUNG FUNCTION BY COMMUNITY, AS A PERCENT OF  
PREDICTED LUNG FUNCTION (%)**

Community	FEV1	FVC	PEFR	MMEF	Percent with FEV1 less than 80% of Predicted	
					FEF <sub>25</sub>	Predicted
Alpine	100.4	101.1	99.8	99.4	99.4	1.4
Lake Elsinore	98.9	99.4	100.1	99.3	98.6	2.4
Lake Arrowhead	100.1	100.3	100.3	100.5	100.2	1.5
Lancaster	99.4	98.9	100.0	99.9	101.7	2.4
Lompoc	100.1	100.4	102.4	100.7	99.5	3.1
Long Beach	99.2	98.8	101.2	101.3	100.2	1.3
Mira Loma	100.0	100.5	97.5	97.3	97.7	1.6
Riverside	98.9	98.9	97.6	98.6	100.2	2.4
San Dimas	99.5	99.1	99.7	99.7	99.7	2.0
Atascadero	102.1	101.9	100.8	100.7	99.1	2.5
Santa Maria	100.3	100.5	99.5	101.9	103.6	2.1
Upland	101.5	100.5	101.6	101.3	100.9	2.1

**TABLE 4.2.2 MORBIDITY PREVALENCE BY COMMUNITY AND GRADE IN SCHOOL  
(QUESTIONNAIRE) (%)**

<b>Community</b>	<b>GRADE</b>	<b>ASTHMA</b>	<b>WHEEZE</b>	<b>BRONCHITIS</b>	<b>COUGH</b>	<b>PNEUMONIA</b>	<b>OTHER CHEST</b>
Alpine	4	5.0	3.4	11.3	5.0	2.1	4.3
	7	8.1	1.6	11.0	6.9	0.0	7.2
	10	11.1	4.8	11.3	9.9	0.0	5.8
	All	7.4	3.3	11.2	6.7	1.0	5.4
Lake Elsinore	4	5.0	5.9	15.5	6.3	2.1	2.9
	7	7.9	11.3	21.3	15.2	1.3	9.2
	10	10.7	5.6	9.2	6.6	0.0	2.7
	All	7.2	7.2	15.4	8.7	1.4	4.5
Lake Arrowhead	4	5.4	6.3	11.2	7.4	2.7	6.0
	7	11.5	2.9	12.5	5.6	2.2	2.3
	10	3.9	7.2	18.2	6.6	2.6	5.2
	All	6.7	5.7	13.5	6.7	2.5	4.8
Lancaster	4	5.8	11.4	12.8	6.5	0.7	7.5
	7	11.5	8.3	12.9	1.6	0.0	1.7
	10	7.3	2.2	12.3	10.3	0.0	3.6
	All	7.5	8.7	12.7	6.2	0.4	5.2
Lompoc	4	4.6	6.1	9.4	4.7	1.8	7.5
	7	11.1	3.0	16.3	11.2	4.9	2.6
	10	4.2	3.8	14.1	5.1	2.0	2.1
	All	6.3	4.5	13.0	6.6	2.8	4.3
Long Beach	4	8.6	7.2	8.3	7.4	3.1	2.0
	7	4.1	1.4	5.3	8.0	0.0	8.2
	10	10.7	1.6	10.7	9.2	1.4	4.2
	All	8.0	4.4	8.1	8.0	1.9	4.3
Mira Loma	4	6.8	3.7	8.9	6.7	1.2	5.1
	7	4.9	5.7	11.3	6.2	2.5	5.0
	10	3.4	2.0	8.6	6.8	0.0	3.4
	All	5.7	3.9	9.5	6.6	1.3	4.7
Riverside	4	10.7	5.8	8.2	6.4	0.6	1.2
	7	11.6	2.5	8.5	3.1	2.1	3.2
	10	9.2	8.2	10.0	5.3	1.4	4.2
	All	10.6	5.4	8.7	5.0	1.2	2.4
San Dimas	4	5.0	4.1	13.2	5.6	2.2	3.6
	7	10.7	6.6	8.0	6.6	0.0	3.9
	10	7.5	0.0	19.7	10.1	2.9	3.0
	All	7.1	3.9	13.4	7.0	1.8	3.6
Atascadero	4	3.8	8.7	20.2	8.0	2.4	4.1
	7	14.5	1.8	14.7	5.6	0.0	5.7
	10	7.3	2.6	19.3	5.2	5.2	5.2
	All	12.6	5.3	18.5	6.7	2.4	4.8
Santa Maria	4	8.8	3.5	8.1	8.1	0.0	3.1
	7	7.1	6.8	7.4	11.1	4.2	5.6
	10	7.9	5.0	16.4	5.3	1.3	9.5
	All	8.2	4.7	10.1	8.1	1.4	5.5
Upland	4	5.7	3.8	19.7	6.3	4.0	4.9
	7	11.9	5.8	15.0	3.4	1.7	3.4
	10	13.6	2.0	10.4	10.4	0.0	6.2
	All	9.3	3.8	16.1	6.8	2.4	4.9

**TABLE 4.3.1.1 UNIVARIATE RELATIONSHIP BETWEEN FEV<sub>1</sub> AND COMMUNITY HISTORICAL  
LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
Peak O <sub>3</sub>	-0.24	(-0.83, 0.35)	0.43
PM <sub>10</sub>	-0.45	(-1.07, 0.17)	0.16
NO <sub>2</sub>	-0.42	(-1.35, 0.51)	0.38
Acid	-5.41	(-20.93, 10.10)	0.49

FEV<sub>1</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, sex, race, height, weight, spirometer, technician, hispanic origin, asthma, pets, and gas stove.

**TABLE 4.3.1.2 UNIVARIATE RELATIONSHIP BETWEEN FVC AND COMMUNITY HISTORICAL  
LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
Peak O <sub>3</sub>	-0.31	(-0.98, 0.36)	0.37
PM <sub>10</sub>	-0.70	(-1.40, -0.01)	0.05
NO <sub>2</sub>	-1.06	(-2.11, -0.01)	0.05
Acid	-20.12	(-37.57, -2.68)	0.02

FVC is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, height, weight, spirometer, technician, hispanic origin and gas stove.



TABLE 4.3.1.3 UNIVARIATE RELATIONSHIP BETWEEN PEFR AND HISTORICAL LEVELS OF AIR POLLUTANTS

Pollutant	Coefficient	95% C.I.	P-value
Peak O <sub>3</sub>	-2.29	(-4.22, -0.36)	0.02
PM <sub>10</sub>	-2.54	(-4.55, -0.53)	0.01
NO <sub>2</sub>	-1.38	(-4.42, 1.67)	0.38
Acid	36.87	(-14.09, 87.82)	0.16

PEFR is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, sex, race, height, weight, spirometer, technician, hispanic origin, asthma, pets, pests, recent exercise, and passive smoke.

TABLE 4.3.1.4 UNIVARIATE RELATIONSHIP BETWEEN MMEF AND HISTORICAL LEVELS OF AIR POLLUTANTS

Pollutant	Coefficient	95% C.I.	P-value
Peak O <sub>3</sub>	-1.37	(-2.73, 0.00)	0.05
PM <sub>10</sub>	-1.30	(-2.73, 0.12)	0.07
NO <sub>2</sub>	-0.47	(-2.57, 1.63)	0.66
Acid	13.54	(-22.02, 49.09)	0.46

MMEF is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, height, weight, spirometer, technician, asthma, hay fever, and passive smoke.

TABLE 4.3.1.5 UNIVARIATE RELATIONSHIP BETWEEN FEF<sub>25</sub> AND HISTORICAL LEVELS OF AIR POLLUTANTS

Pollutant	Coefficient	95% C.I.	P-value
Peak O <sub>3</sub>	-0.70	(-1.99, 0.59)	0.29
PM <sub>10</sub>	-0.50	(-1.85, 0.85)	0.47
NO <sub>2</sub>	-0.35	(-2.34, 1.65)	0.73
Acid	8.54	(-25.19, 42.27)	0.62

FEF<sub>25</sub> is measured in ml/sec, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, height, weight, spirometer, technician, asthma, and hay fever.

TABLE 4.3.1.6 MULTIVARIATE RELATIONSHIP BETWEEN PULMONARY FUNCTION MEASUREMENTS AND HISTORICAL LEVELS OF AIR POLLUTANTS

Pollutant	Coefficient	95% C.I.	P-value
<b>FVC</b>			
PM <sub>10</sub>	-0.63	(-1.71, 0.44)	0.25
NO <sub>2</sub>	0.24	(-1.61, 2.10)	0.80
Acid	-18.51	(-40.18, 3.16)	0.09
<b>PEFR</b>			
Peak O <sub>3</sub>	-0.89	(-4.20, 2.42)	0.60
PM <sub>10</sub>	-1.79	(-5.24, 1.66)	0.31
<b>MMEF</b>			
Peak O <sub>3</sub>	-1.04	(-3.38, 1.30)	0.38
PM <sub>10</sub>	-0.42	(-2.86, 2.02)	0.73

O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, height, weight, spirometer, technician, asthma, hay fever, and passive smoke.

**TABLE 4.3.2.1 UNIVARIATE RELATIONSHIP BETWEEN FEV<sub>1</sub> AND LIFETIME EXPOSURE TO AIR  
POLLUTANTS  
BY GRADE AND BY GENDER**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>Peak O<sub>3</sub></b>			
4th grade	-0.75	(-2.34, 0.84)	0.36
7th grade	0.32	(-2.55, 3.18)	0.83
10th grade	-3.42	(-6.98, 0.15)	0.06
Boys	-3.22	(-5.47, -0.96)	0.01
Girls	0.63	(-1.19, 2.44)	0.50
<b>ALL</b>	<b>-1.25</b>	<b>(-2.71, 0.22)</b>	<b>0.10</b>
<b>PM<sub>10</sub></b>			
4th grade	0.00	(-2.36, 2.36)	1.00
7th grade	2.38	(-2.32, 7.08)	0.32
10th grade	-3.19	(-9.32, 2.94)	0.31
Boys	0.04	(-3.60, 3.68)	0.98
Girls	-0.28	(-3.13, 2.56)	0.85
<b>ALL</b>	<b>0.05</b>	<b>(-2.27, 2.37)</b>	<b>0.97</b>
<b>NO<sub>2</sub></b>			
4th grade	-0.05	(-2.92, 2.82)	0.97
7th grade	3.17	(1.93, 8.27)	0.22
10th grade	-1.66	(-8.03, 4.71)	0.61
Boys	-0.08	(-4.31, 4.14)	0.97
Girls	0.50	(-2.69, 3.68)	0.76
<b>ALL</b>	<b>0.22</b>	<b>(-2.42, 2.86)</b>	<b>0.87</b>
<b>AVG O<sub>3</sub></b>			
4th grade	-0.91	(-4.05, 2.23)	0.57
7th grade	-2.76	(-8.63, 3.11)	0.36
10th grade	-5.53	(-13.50, 2.43)	0.17
Boys	-6.43	(-10.89, -1.96)	0.00
Girls	1.53	(-2.34, 5.41)	0.44
<b>ALL</b>	<b>-2.80</b>	<b>(-5.81, 0.21)</b>	<b>0.07</b>

FEV<sub>1</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, spirometer, technician, hispanic origin, asthma, pets, and gas stove. Results for 4th, 7th and 10th grades also adjusted for sex.

TABLE 4.3.2.2.1 UNIVARIATE RELATIONSHIP BETWEEN FVC AND LIFETIME EXPOSURE TO AIR POLLUTANTS

Pollutant	Coefficient	95% C.I.	P-value
Peak O <sub>3</sub>	-0.77	(-2.43, 0.89)	0.36
PM <sub>10</sub>	-0.66	(-3.27, 1.94)	0.62
NO <sub>2</sub>	-1.97	(-4.92, 0.99)	0.19
AVG O <sub>3</sub>	0.06	(-3.35, 3.48)	0.97

FVC is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, spirometer, technician, hispanic origin, and gas stove.

TABLE 4.3.2.2.2 UNIVARIATE RELATIONSHIP BETWEEN FVC AND LIFETIME EXPOSURE TO AIR POLLUTANTS BY GRADE AND BY GENDER

Pollutant	Coefficient	95% C.I.	P-value
Peak O <sub>3</sub>			
4th grade	0.19	(-1.68, 2.05)	0.84
7th grade	0.36	(-2.84, 3.56)	0.83
10th grade	-2.76	(-6.79, 1.27)	0.18
Boys	-2.31	(-4.84, 0.23)	0.07
Girls	0.95	(-1.08, 2.98)	0.36
PM <sub>10</sub>			
4th grade	0.56	(-2.20, 3.33)	0.69
7th grade	-0.46	(-5.68, 4.77)	0.86
10th grade	-3.50	(-10.24, 3.24)	0.31
Boys	0.22	(-3.85, 4.29)	0.92
Girls	-1.41	(-4.55, 1.74)	0.38
NO <sub>2</sub>			
4th grade	-1.94	(-5.27, 1.39)	0.25
7th grade	0.56	(-5.05, 6.17)	0.84
10th grade	-4.53	(-11.66, 2.61)	0.21
Boys	-0.73	(-5.38, 3.92)	0.76
Girls	-2.60	(-6.15, 0.94)	0.15
AVG O <sub>3</sub>			
4th grade	2.67	(-1.04, 6.38)	0.16
7th grade	-0.16	(-6.70, 6.37)	0.96
10th grade	-1.49	(-10.51, 7.53)	0.75
Boys	-3.41	(-8.46, 1.65)	0.19
Girls	4.29	(-0.05, 8.62)	0.05

FVC is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, spirometer, technician, hispanic origin, and gas stove. Results for 4th, 7th and 10th grades also adjusted for sex.

**TABLE 4.3.2.3**  
**UNIVARIATE RELATIONSHIP BETWEEN PEFR AND LIFETIME EXPOSURE TO AIR POLLUTANTS**  
**BY GRADE AND BY GENDER**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>Peak O<sub>3</sub></b>			
4th grade	-3.76	(-9.24, 1.72)	0.18
7th grade	1.30	(-8.28, 10.88)	0.79
10th grade	-9.94	(-21.81, 1.92)	0.10
Boys	-9.78	(-17.00, -2.57)	0.01
Girls	-0.62	(-6.74, 5.50)	0.84
<b>ALL</b>	<b>-4.92</b>	<b>(-9.67, -0.18)</b>	<b>0.04</b>
<b>PM<sub>10</sub></b>			
4th grade	5.65	(-2.48, 13.78)	0.17
7th grade	10.73	(-4.83, 26.28)	0.18
10th grade	-19.91	(-39.17, -0.66)	0.04
Boys	-6.73	(-18.01, 4.55)	0.24
Girls	6.45	(-3.12, 16.02)	0.19
<b>ALL</b>	<b>1.21</b>	<b>(-6.18, 8.60)</b>	<b>0.75</b>
<b>NO<sub>2</sub></b>			
4th grade	4.26	(-5.67, 14.20)	0.40
7th grade	24.76	(7.76, 41.76)	0.00
10th grade	5.41	(-14.65, 25.47)	0.60
Boys	3.46	(-9.69, 16.61)	0.61
Girls	14.39	(3.73, 25.05)	0.01
<b>ALL</b>	<b>9.29</b>	<b>(0.87, 17.70)</b>	<b>0.03</b>
<b>AVG O<sub>3</sub></b>			
4th grade	-12.41	(-23.22, -1.61)	0.02
7th grade	-14.25	(-34.01, 5.51)	0.16
10th grade	-18.80	(-45.37, 7.77)	0.17
Boys	-20.82	(-35.21, -6.43)	0.00
Girls	-11.63	(-24.62, 1.35)	0.08
<b>ALL</b>	<b>-16.91</b>	<b>(-26.64, -7.17)</b>	<b>0.00</b>

PEFR is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, spirometer, technician, hispanic origin, asthma, pets, pests, recent exercise, and passive smoke. Results for 4th, 7th and 10th grades also adjusted for sex.

**TABLE 4.3.2.4 UNIVARIATE RELATIONSHIP BETWEEN MMEF AND LIFETIME EXPOSURE TO AIR  
POLLUTANTS  
BY GRADE AND GENDER**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>Peak O<sub>3</sub></b>			
4th grade	-0.58	(-4.66, 3.50)	0.78
7th grade	-1.27	(-7.67, 5.44)	0.71
10th grade	-6.37	(-14.43, 1.68)	0.12
Boys	-6.72	(-11.69, -1.76)	0.01
Girls	1.03	(-3.40, 5.46)	0.65
<b>ALL</b>	<b>-2.63</b>	<b>(-5.93, 0.67)</b>	<b>0.12</b>
<b>PM<sub>10</sub></b>			
4th grade	1.77	(-4.35, 7.89)	0.57
7th grade	11.15	(0.19, 22.10)	0.05
10th grade	-4.29	(-18.27, 9.70)	0.55
Boys	3.50	(-11.53, 4.52)	0.39
Girls	6.21	(-0.84, 13.26)	0.08
<b>ALL</b>	<b>1.73</b>	<b>(-3.56, 7.01)</b>	<b>0.52</b>
<b>NO<sub>2</sub></b>			
4th grade	5.10	(-2.44, 12.64)	0.19
7th grade	15.09	(3.07, 27.10)	0.01
10th grade	10.98	(-3.44, 25.41)	0.14
Boys	3.49	(-5.89, 12.87)	0.47
Girls	11.48	(3.61, 19.35)	0.00
<b>ALL</b>	<b>7.92</b>	<b>(1.89, 13.95)</b>	<b>0.01</b>
<b>AVG O<sub>3</sub></b>			
4th grade	-7.51	(-15.83, 0.82)	0.08
7th grade	-15.09	(-28.69, -1.48)	0.03
10th grade	-20.35	(-38.57, -2.14)	0.03
Boys	-16.30	(-26.20, -6.40)	0.00
Girls	-6.45	(-16.12, 3.22)	0.19
<b>ALL</b>	<b>-11.76</b>	<b>(-18.65, -4.88)</b>	<b>0.00</b>

MMEF is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, asthma, spirometer, technician, hayfever, and passive smoke. Results for 4th, 7th, and 10th grades also adjusted for sex.

**TABLE 4.3.2.5 UNIVARIATE RELATIONSHIP BETWEEN FEF<sub>25</sub> AND LIFETIME EXPOSURE  
TO AIR POLLUTANTS  
BY GRADE AND GENDER**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>Peak O<sub>3</sub></b>			
4th grade	-0.10	(-3.94, 3.74)	0.96
7th grade	-3.43	(-10.05, 3.19)	0.31
10th grade	-3.43	(-11.11, 4.25)	0.38
Boys	-4.98	(-9.71, -0.25)	0.04
Girls	0.73	(-3.61, 5.06)	0.74
<b>ALL</b>	<b>-1.88</b>	<b>(-5.06, 1.30)</b>	<b>0.25</b>
<b>PM<sub>10</sub></b>			
4th grade	1.65	(-4.13, 7.42)	0.58
7th grade	7.37	(-3.45, 18.19)	0.18
10th grade	-3.70	(-16.87, 9.47)	0.58
Boys	-4.37	(-11.93, 3.19)	0.26
Girls	5.35	(-1.58, 12.27)	0.13
<b>ALL</b>	<b>0.90</b>	<b>(-4.17, 5.96)</b>	<b>0.73</b>
<b>NO<sub>2</sub></b>			
4th grade	5.05	(-2.00, 12.11)	0.16
7th grade	13.27	(1.19, 25.34)	0.03
10th grade	8.98	(-4.65, 22.61)	0.20
Boys	3.12	(-5.72, 11.97)	0.49
Girls	10.89	(3.15, 18.63)	0.01
<b>ALL</b>	<b>7.45</b>	<b>(1.66, 13.24)</b>	<b>0.01</b>
<b>AVG O<sub>3</sub></b>			
4th grade	-7.46	(-15.30, 0.38)	0.06
7th grade	-18.75	(-32.31, -5.19)	0.01
10th grade	-12.01	(-29.24, 5.23)	0.17
Boys	-12.12	(-21.52, -2.71)	0.01
Girls	-8.76	(-18.23, 0.70)	0.07
<b>ALL</b>	<b>-10.42</b>	<b>(-17.03, -3.80)</b>	<b>0.00</b>

O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for age, race, height, weight, spirometer, technician, asthma, hay fever, and passive smoke. Grades also adjusted for sex. FEF<sub>25</sub> is measured in ml/sec.

**TABLE 4.3.3.1 PULMONARY FUNCTION BY PAST INDIVIDUALLY CONSTRUCTED EXPOSURE HISTORY (MIGRANTS ONLY)**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>FEV<sub>1</sub></b>			
PEAK O <sub>3</sub>	-1.47	(-3.09 , 0.15)	0.08
PM <sub>10</sub>	-0.69	(-3.23 , 1.86)	0.60
NO <sub>2</sub>	0.70	(-2.19 , 3.59)	0.64
AVG O <sub>3</sub>	-4.15	(-7.81 , -0.49)	0.03
<b>FVC</b>			
PEAK O <sub>3</sub>	-1.06	(-2.90, 0.78)	0.26
PM <sub>10</sub>	-1.33	(-4.19, 1.53)	0.36
NO <sub>2</sub>	-0.97	(-4.21, 2.27)	0.56
AVG O <sub>3</sub>	-0.85	(-5.03, 3.32)	0.69
<b>PEFR</b>			
PEAK O <sub>3</sub>	-5.46	(-10.84, -0.08)	0.05
PM <sub>10</sub>	-0.42	(-8.67, 7.83)	0.92
NO <sub>2</sub>	10.10	(0.71, 19.48)	0.04
AVG O <sub>3</sub>	-21.81	(-33.97, -9.65)	0.00
<b>MMEF</b>			
PEAK O <sub>3</sub>	-3.60	(-7.36, 0.17)	0.06
PM <sub>10</sub>	0.47	(-5.49, 6.43)	0.88
NO <sub>2</sub>	8.30	(1.52, 15.07)	0.02
AVG O <sub>3</sub>	-17.76	(-26.35, -9.17)	0.00
<b>FEF<sub>25</sub></b>			
PEAK O <sub>3</sub>	-2.12	(-5.73, 1.50)	0.25
PM <sub>10</sub>	0.08	(-5.62, 5.78)	0.98
NO <sub>2</sub>	7.05	(0.56, 13.54)	0.03
AVG O <sub>3</sub>	-13.93	(-22.18, -5.68)	0.00

Units and adjustments are as described in section 4.3.2.



**TABLE 4.3.4.1 RELATIONSHIP BETWEEN FVC AND LIFETIME EXPOSURE TO AIR POLLUTANTS, ADJUSTED FOR PHYSICAL ACTIVITY**

Pollutant	Coefficient	95% C.I.	P-value
PEAK O <sub>3</sub>	-1.15	(-2.93 , 0.62)	0.20
PM <sub>10</sub>	-0.79	(-3.61 , 2.03)	0.58
NO <sub>2</sub>	-2.39	(-5.55 , 0.77)	0.14
AVG O <sub>3</sub>	-0.33	(-3.98 , 3.33)	0.86

FVC is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, weight, spirometer, technician, hispanic origin, pets, gas stove, and number of hours spent outdoors during two summer weeks.

**TABLE 4.3.4.2 RELATIONSHIP BETWEEN PEFR AND LIFETIME EXPOSURE TO AIR POLLUTANTS, ADJUSTED FOR PHYSICAL ACTIVITY**

Pollutant	Coefficient	95% C.I.	P-value
PEAK O <sub>3</sub>	-5.35	(-10.39 , -0.30)	0.04
PM <sub>10</sub>	-0.30	(-8.23 , 7.64)	0.94
NO <sub>2</sub>	8.48	(-8.46 , 17.42)	0.06
AVG O <sub>3</sub>	-17.76	(-28.12 , -7.41)	0.00

PEFR is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, height, weight, spirometer, technician, asthma, pets, and number of hours spent outdoors during two summer weeks.

**TABLE 4.4.1 RELATIONSHIP BETWEEN FEV<sub>1</sub> MEASURED IN 1994 AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
Peak O <sub>3</sub>	-0.77	(-2.55, 1.01)	0.40
PM <sub>10</sub>	1.95	(-0.94, 4.84)	0.19
NO <sub>2</sub>	0.40	(-2.82, 3.63)	0.81
AVG O <sub>3</sub>	-2.47	(-6.08, 1.15)	0.18

FEV<sub>1</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, sex, race, height, weight, hispanic origin, asthma, pets, and gas stove.

**TABLE 4.4.2 RELATIONSHIP BETWEEN FVC MEASURED IN 1994 AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Coefficient</b>	<b>95% C.I.</b>	<b>P-value</b>
Peak O <sub>3</sub>	-1.06	(-3.01, 0.88)	0.28
PM <sub>10</sub>	0.39	(-2.76, 3.55)	0.81
NO <sub>2</sub>	-2.14	(-5.64, 1.36)	0.23
AVG O <sub>3</sub>	-1.24	(-5.21, 2.72)	0.54

FVC<sub>1</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, sex, race, height, weight, hispanic origin, asthma, pets, and gas stove.

**TABLE 4.5.1.1 RELATIONSHIP BETWEEN ASTHMA PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Ozone	1.00	(0.99, 1.01)	0.92
PM <sub>10</sub>	1.01	(1.00, 1.01)	0.15
NO <sub>2</sub>	1.00	(0.99, 1.02)	0.55
Acid	0.99	(0.82, 1.20)	0.93

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, hayfever, health insurance, houseplants, pests, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.1.2 RELATIONSHIP BETWEEN WHEEZE PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	1.00	(0.99, 1.01)	0.85
PM <sub>10</sub>	1.00	(0.99, 1.01)	0.85
NO <sub>2</sub>	0.99	(0.98, 1.01)	0.27
Acid	1.07	(0.85, 1.35)	0.55

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, and hayfever. Relative risk is per unit of exposure.

**TABLE 4.5.1.3 RELATIONSHIP BETWEEN BRONCHITIS PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
Ozone	1.00	(1.00, 1.01)	0.47
PM <sub>10</sub>	1.00	(0.99, 1.00)	0.48
NO <sub>2</sub>	1.00	(0.99, 1.00)	0.30
Acid	1.07	(0.92, 1.24)	0.24

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, hayfever, houseplants, pests, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.1.4 RELATIONSHIP BETWEEN COUGH PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
Ozone	1.00	(0.99, 1.01)	0.90
PM <sub>10</sub>	1.00	(0.99, 1.01)	0.99
NO <sub>2</sub>	1.00	(0.98, 1.01)	0.58
Acid	1.00	(0.79, 1.26)	0.99

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, hayfever, vitamins, income, mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.1.5 RELATIONSHIP BETWEEN PNEUMONIA PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Ozone	1.00	(0.99, 1.01)	0.92
PM <sub>10</sub>	1.00	(0.99, 1.01)	0.96
NO <sub>2</sub>	1.00	(0.99, 1.02)	0.65
Acid	0.88	(0.64, 1.23)	0.46

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, and health insurance. Relative risk is per unit of exposure.

**TABLE 4.5.1.6 RELATIONSHIP BETWEEN OTHER CHEST ILLNESS PREVALENCE AND HISTORICAL LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Ozone	0.98	(0.72, 1.35)	0.91
PM <sub>10</sub>	0.98	(0.89, 1.09)	0.77
NO <sub>2</sub>	0.94	(0.87, 1.02)	0.15
Acid	1.33	(0.82, 2.15)	0.24

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb, and acid is HCl + HNO<sub>3</sub> measured in ppb on a mole basis. Adjusted for age, race, sex, hayfever, vitamins, income, mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.2.1.1 RELATIONSHIP BETWEEN ASTHMA PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	1.02	(1.00, 1.04)	0.07
PM <sub>10</sub>	1.01	(0.99, 1.04)	0.31
NO <sub>2</sub>	1.02	(0.99, 1.05)	0.18
AVG O <sub>3</sub>	1.01	(0.97, 1.05)	0.54

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, hayfever, health insurance, houseplants, pests, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.2.1.2 RELATIONSHIP BETWEEN ASTHMA PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS BY GENDER**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>			
Boys	1.02	(0.99, 1.05)	0.20
Girls	1.02	(0.99, 1.05)	0.19
PM <sub>10</sub>			
Boys	1.01	(0.97, 1.05)	0.61
Girls	1.02	(0.98, 1.07)	0.30
NO <sub>2</sub>			
Boys	1.02	(0.97, 1.07)	0.50
Girls	1.02	(0.98, 1.07)	0.30
AVG O <sub>3</sub>			
Boys	1.01	(0.96, 1.07)	0.65
Girls	1.01	(0.95, 1.08)	0.68

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, hayfever, health insurance, houseplants, pests, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.2.2.1 RELATIONSHIP BETWEEN WHEEZE PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	1.00	(0.97, 1.02)	0.81
PM <sub>10</sub>	1.02	(0.98, 1.05)	0.42
NO <sub>2</sub>	0.99	(0.95, 1.03)	0.58
AVG O <sub>3</sub>	0.99	(0.94, 1.04)	0.74

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, and hayfever. Relative risk is per unit of exposure.

**TABLE 4.5.2.2.2 RELATIONSHIP BETWEEN WHEEZE PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS STRATIFIED BY GENDER**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>			
Boys	1.00	(0.99, 1.02)	0.85
Girls	1.00	(0.98, 1.01)	0.65
PM <sub>10</sub>			
Boys	1.00	(0.98, 1.03)	0.86
Girls	1.01	(0.98, 1.03)	0.67
NO <sub>2</sub>			
Boys	0.99	(0.96, 1.01)	0.31
Girls	0.98	(0.96, 1.01)	0.25
AVG O <sub>3</sub>			
Boys	1.01	(0.97, 1.05)	0.70
Girls	0.99	(0.95, 1.03)	0.57

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, hayfever, health insurance, houseplants, mold or mildew, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.2.3.1 RELATIONSHIP BETWEEN BRONCHITIS PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
PEAK O <sub>3</sub>	1.00	(0.99, 1.02)	0.83
PM <sub>10</sub>	1.03	(1.00, 1.05)	0.02
NO <sub>2</sub>	1.00	(0.97, 1.02)	0.81
AVG O <sub>3</sub>	0.99	(0.96, 1.02)	0.68

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, hay fever, mold or mildew, and carpet. Relative risk is per unit of exposure.

**TABLE 4.5.2.3.2 RELATIONSHIP BETWEEN BRONCHITIS PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS STRATIFIED BY GENDER**

Pollutant	OR	95% C.I.	P-value
PEAK O <sub>3</sub>			
Boys	1.01	(0.99, 1.03)	0.47
Girls	1.00	(0.98, 1.01)	0.67
PM <sub>10</sub>			
Boys	1.01	(0.98, 1.05)	0.50
Girls	1.04	(1.01, 1.07)	0.01
NO <sub>2</sub>			
Boys	0.99	(0.95, 1.03)	0.75
Girls	1.00	(0.97, 1.03)	0.95
AVG O <sub>3</sub>			
Boys	1.01	(0.96, 1.05)	0.70
Girls	0.98	(0.94, 1.02)	0.28

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, hay fever, mold or mildew, and carpet. Relative risk is per unit of exposure.



**TABLE 4.5.2.4.1 RELATIONSHIP BETWEEN COUGH PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
PEAK O <sub>3</sub>	1.01	(0.98, 1.03)	0.53
PM <sub>10</sub>	1.02	(0.99, 1.06)	0.25
NO <sub>2</sub>	0.99	(0.94, 1.03)	0.48
AVG O <sub>3</sub>	1.00	(0.96, 1.05)	0.87

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, height, hay fever, vitamins, income, and mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.2.4.2 RELATIONSHIP BETWEEN COUGH PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS STRATIFIED BY GENDER**

Pollutant	OR	95% C.I.	P-value
PEAK O <sub>3</sub>			
Boys	1.01	(0.98, 1.04)	0.63
Girls	1.01	(0.98, 1.04)	0.60
PM <sub>10</sub>			
Boys	1.00	(0.96, 1.06)	0.84
Girls	1.04	(0.99, 1.09)	0.13
NO <sub>2</sub>			
Boys	0.99	(0.93, 1.05)	0.67
Girls	0.99	(0.93, 1.05)	0.68
AVG O <sub>3</sub>			
Boys	1.01	(0.95, 1.08)	0.70
Girls	0.99	(0.92, 1.06)	0.76

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, height, hay fever, vitamins, income, and mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.2.5.1 RELATIONSHIP BETWEEN PNEUMONIA PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	0.99	(0.98, 11.00)	0.14
PM <sub>10</sub>	1.00	(0.97, 1.02)	0.69
NO <sub>2</sub>	1.00	(0.98, 1.03)	0.75
AVG O <sub>3</sub>	0.94	(0.90, 0.99)	0.01

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for health insurance. Relative risk is per unit of exposure.

**TABLE 4.5.2.5.2 RELATIONSHIP BETWEEN PNEUMONIA PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS STRATIFIED BY GENDER**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>			
Boys	0.99	(0.96, 1.01)	0.16
Girls	0.99	(0.98, 1.01)	0.53
PM <sub>10</sub>			
Boys	1.00	(0.96, 1.03)	0.86
Girls	0.99	(0.96, 1.02)	0.69
NO <sub>2</sub>			
Boys	1.00	(0.97, 1.04)	0.81
Girls	1.00	(0.97, 1.04)	0.81
AVG O <sub>3</sub>			
Boys	0.91	(0.85, 0.98)	0.01
Girls	0.97	(0.92, 1.03)	0.31

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Relative risk is per unit of exposure.

**TABLE 4.5.2.6.1 RELATIONSHIP BETWEEN OTHER CHEST ILLNESS PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	1.01	(0.99, 1.03)	0.55
PM <sub>10</sub>	0.99	(0.96, 1.03)	0.71
NO <sub>2</sub>	1.00	(0.96, 1.04)	0.97
AVG O <sub>3</sub>	1.01	(0.97, 1.06)	0.61

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, vitamins, and mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.2.6.2 RELATIONSHIP BETWEEN OTHER CHEST ILLNESS PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS, STRATIFIED BY GENDER**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>			
BOYS	1.00	(0.97, 1.03)	0.93
GIRLS	1.02	(0.98, 1.05)	0.35
PM <sub>10</sub>			
BOYS	0.99	(0.94, 1.04)	0.75
GIRLS	1.00	(0.95, 1.05)	0.89
NO <sub>2</sub>			
BOYS	1.01	(0.96, 1.07)	0.63
GIRLS	0.99	(0.94, 1.04)	0.65
AVG O <sub>3</sub>			
BOYS	0.99	(0.93, 1.05)	0.74
GIRLS	1.04	(0.97, 1.12)	0.28

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, hay fever, vitamins, and mold or mildew. Relative risk is per unit of exposure.

**TABLE 4.5.3.1 RELATIONSHIP BETWEEN DISEASE PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS: MIGRANTS ONLY**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
<b>Asthma</b>			
PEAK O <sub>3</sub>	1.02	(1.00, 1.04)	0.08
PM <sub>10</sub>	1.01	(0.98, 1.04)	0.51
NO <sub>2</sub>	1.01	(0.98, 1.04)	0.63
AVG O <sub>3</sub>	1.03	(0.98, 1.09)	0.19
<b>Wheeze</b>			
PEAK O <sub>3</sub>	1.00	(0.99, 1.02)	0.62
PM <sub>10</sub>	1.01	(0.98, 1.03)	0.62
NO <sub>2</sub>	0.99	(0.97, 1.01)	0.37
AVG O <sub>3</sub>	1.02	(0.98, 1.06)	0.42
<b>Bronchitis</b>			
PEAK O <sub>3</sub>	1.00	(0.99, 1.02)	0.68
PM <sub>10</sub>	1.03	(1.00, 1.05)	0.03
NO <sub>2</sub>	0.99	(0.96, 1.02)	0.52
AVG O <sub>3</sub>	1.00	(0.97, 1.04)	0.85
<b>Cough</b>			
PEAK O <sub>3</sub>	1.00	(0.98, 1.03)	0.80
PM <sub>10</sub>	1.02	(0.99, 1.06)	0.23
NO <sub>2</sub>	0.98	(0.94, 1.03)	0.50
AVG O <sub>3</sub>	1.00	(0.95, 1.06)	0.95
<b>Pneumonia</b>			
PEAK O <sub>3</sub>	0.98	(0.96, 1.00)	0.06
PM <sub>10</sub>	0.98	(0.95, 1.01)	0.19
NO <sub>2</sub>	0.99	(0.96, 1.02)	0.67
AVG O <sub>3</sub>	0.94	(0.88, 1.00)	0.05
<b>Other</b>			
PEAK O <sub>3</sub>	1.01	(0.99, 1.03)	0.41
PM <sub>10</sub>	0.99	(0.95, 1.03)	0.64
NO <sub>2</sub>	0.97	(0.93, 1.01)	0.21
AVG O <sub>3</sub>	1.04	(0.99, 1.10)	0.11

Units and adjustments are as described in section 4.5.2.

**TABLE 4.5.4.1 RELATIONSHIP BETWEEN COUGH PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS, ADJUSTED FOR PHYSICAL ACTIVITY**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	1.01	(0.99, 1.04)	0.38
PM <sub>10</sub>	1.02	(0.99, 1.06)	0.21
NO <sub>2</sub>	0.98	(0.94, 1.03)	0.45
AVG O <sub>3</sub>	1.01	(0.97, 1.06)	0.62

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for community, age, race, sex, hay fever, height, vitamins, income, mold or mildew, and number of hours physically active during the last ten weekdays. Relative risk is per unit of exposure.

**TABLE 4.5.4.2 RELATIONSHIP BETWEEN PNEUMONIA PREVALENCE AND LIFETIME EXPOSURE TO AIR POLLUTANTS, ADJUSTED FOR PHYSICAL ACTIVITY**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	0.99	(0.97, 1.00)	0.14
PM <sub>10</sub>	1.00	(0.97, 1.02)	0.84
NO <sub>2</sub>	1.01	(0.98, 1.03)	0.64
AVG O <sub>3</sub>	0.94	(0.90, 0.99)	0.03

O<sub>3</sub> is measured in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Adjusted for health insurance and the number of hours spent outdoors during the last two weekends.

**Table 4.6.1.1 ABSENCE DATA BY SEX, RACE, AND GRADE**

	Subject	Abs	Interviewed	Proportion Interviewed	Illnesses	Respiratory	Proportion Respiratory
<b>Total</b>	<b>2681</b>	<b>15,658</b>	<b>2,394</b>	<b>0.15</b>	<b>2,334</b>	<b>915</b>	<b>0.39</b>
<b>SEX</b>							
Male	1382	8417	1264	0.15	1239	477	0.38
Female	1283	7170	1121	0.16	1086	433	0.40
<b>RACE</b>							
White	1955	11331	1811	0.16	1762	682	0.39
Black	105	619	53	0.09	51	25	0.49
Asian	99	428	37	0.09	37	17	0.46
Indian	27	220	22	0.10	21	9	0.43
Other	368	2290	372	0.16	365	145	0.40
Mixed	127	770	99	0.13	98	37	0.38
<b>GRADE</b>							
Grade 6	1296	6313	1342	0.21	1311	542	0.41
Grade 9	698	4496	586	0.13	569	221	0.39
Grade 12	629	4620	433	0.09	423	144	0.34
Other	58	229	33	0.14	31	8	0.26

Subjects are those with at least one absence. Sex is unknown for 16 subjects.

**Table 4.6.1.2 ABSENCE DATA BY COMMUNITY**

	Subjects	Abs	Interviewed	Proportion Interviewed	Illnesses	Respiratory	Proportion Respiratory
Alpine	226	1409	219	0.16	211	83	0.39
Lake Elsinore	251	1822	242	0.13	226	88	0.39
Lake Arrowhead	264	1663	156	0.09	155	63	0.41
Lancaster	180	1023	185	0.18	181	69	0.38
Lompoc	239	1582	161	0.10	156	67	0.43
Long Beach	235	1151	74	0.06	71	17	0.24
Mira Loma	228	1261	295	0.23	290	106	0.37
Riverside	231	1365	129	0.09	129	57	0.44
San Dimas	219	1295	460	0.36	452	175	0.39
Atascadero	206	1154	158	0.14	153	59	0.39
Santa Maria	192	870	122	0.14	118	40	0.34
Upland	210	1063	193	0.18	192	91	0.47

Subjects are those with at least one absence.

**Table 4.6.1.3 ABSENCE DATA BY MONTH**

	Subjects	Interviewed	Proportion Interviewed	Illnesses	Respiratory	Proportion Respiratory
January	1432	145	0.10	142	67	0.47
February	1587	290	0.18	283	131	0.46
March	1707	191	0.11	181	71	0.39
April	1529	199	0.13	195	63	0.32
May	1943	477	0.25	467	139	0.30
June	606	219	0.36	218	41	0.19
July	84	25	0.30	25	11	0.44
August	98	17	0.17	17	6	0.35
September	1205	88	0.07	87	25	0.29
October	1934	136	0.07	134	62	0.46
November	2106	384	0.18	368	190	0.52
December	1427	223	0.16	217	109	0.50

Subjects are those with at least one absence.

**Table 4.6.1.4 ABSENCE DATA BY DAY OF WEEK**

	Abs	Interviewed	Proportion Interviewed	Illnesses	Respiratory	Proportion Respiratory
Monday	4309	718	0.17	703	307	0.44
Tuesday	3136	500	0.16	489	196	0.40
Wednesday	2798	399	0.14	387	145	0.37
Thursday	2592	375	0.14	364	151	0.41
Friday	2823	402	0.14	391	116	0.30

**Table 4.6.1.5 ABSENCE DATA BY ASTHMATIC OR WHEEZY STATUS**

	Subjects	Abs.	Absences per Subject	Interviewed	Proportion Interviewed	Illnesses	Respiratory	Proportion Respiratory
Asthmatic	204	1333	6.53	206	0.15	199	106	0.53
Wheezy	295	2060	6.98	327	0.16	321	115	0.36
Healthy	2182	12265	5.62	1861	0.15	1814	694	0.38

Subjects are those with at least one absence.



**TABLE 4.6.2.1 RELATIVE RISK OF ABSENCE ( ALL CASES) IN RELATION TO RECENT EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Relative Risk per Unit* Exposure</b>	<b>95% C.I.</b>
PEAK O <sub>3</sub> lag 1	0.99	(0.98, 1.01)
PEAK O <sub>3</sub> lag 2	1.00	(0.99, 1.02)
PEAK PM <sub>10</sub> lag 1	1.00	(1.00, 1.01)
PEAK PM <sub>10</sub> lag 2	0.99	(0.99, 1.00)
PEAK NO <sub>2</sub> lag 1	1.01	(0.99, 1.03)
PEAK NO <sub>2</sub> lag 2	1.03	(1.01, 1.05)

There were 2491 subjects for whom absences were reported. Adjusted for temperature and humidity lagged one and two days. \*Units are 10 ppb for O<sub>3</sub> and NO<sub>2</sub>, 10 µg/m<sup>3</sup> for PM<sub>10</sub>.

**TABLE 4.6.2.2 RELATIVE RISK OF ABSENCE DUE TO RESPIRATORY ILLNESS IN RELATION TO RECENT EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Relative Risk per Unit* Exposure</b>	<b>95% C.I.</b>
PEAK O <sub>3</sub> lag 1	1.01	(0.95, 1.08)
PEAK O <sub>3</sub> lag 2	1.00	(0.94, 1.07)
PEAK PM <sub>10</sub> lag 1	0.97	(0.93, 1.00)
PEAK PM <sub>10</sub> lag 2	1.00	(0.96, 1.03)
PEAK NO <sub>2</sub> lag 1	1.02	(0.95, 1.09)
PEAK NO <sub>2</sub> lag 2	1.00	(0.93, 1.08)

There were 492 subjects who reported an absence due to respiratory illness. Adjusted for temperature humidity lagged one and two days. \*Units are 10 ppb for O<sub>3</sub> and NO<sub>2</sub>, 10 µg/m<sup>3</sup> for PM<sub>10</sub>.

**TABLE 4.6.2.3 RELATIVE RISK OF ABSENCE DUE TO RESPIRATORY ILLNESS IN RELATION TO RECENT EXPOSURE TO AIR POLLUTANTS: WHEEZERS ONLY**

<b>Pollutant</b>	<b>Relative Risk per Unit Exposure</b>	<b>95% C.I.</b>
PEAK O <sub>3</sub> lag 1	1.03	(0.74, 1.43)
PEAK O <sub>3</sub> lag 2	1.11	(0.71, 1.73)
PEAK PM <sub>10</sub> lag 1	0.83	(0.60, 1.16)
PEAK PM <sub>10</sub> lag 2	0.68	(0.43, 1.08)
PEAK NO <sub>2</sub> lag 1	1.26	(0.97, 1.65)
PEAK NO <sub>2</sub> lag 2	1.38	(0.82, 2.30)

There were 60 wheezers who reported an absence due to respiratory illness. Adjusted for temperature and humidity lagged one and two days. Units are 10 ppb for O<sub>3</sub> and NO<sub>2</sub>, 10 µg/m<sup>3</sup> for PM<sub>10</sub>.

**TABLE 4.6.2.4 RELATIVE RISK OF ABSENCE DUE TO RESPIRATORY ILLNESS IN RELATION TO RECENT EXPOSURE TO AIR POLLUTANTS: ASTHMATICS ONLY**

<b>Pollutant</b>	<b>Relative Risk per Unit Exposure</b>	<b>95% C.I.</b>
PEAK O <sub>3</sub> lag 1	0.95	(0.73, 1.24)
PEAK O <sub>3</sub> lag 2	1.10	(0.80, 1.53)
PEAK PM <sub>10</sub> lag 1	0.92	(0.76, 1.12)
PEAK PM <sub>10</sub> lag 2	0.94	(0.80, 1.10)
PEAK NO <sub>2</sub> lag 1	0.88	(0.69, 1.13)
PEAK NO <sub>2</sub> lag 2	0.89	(0.62, 1.28)

There were 55 asthmatics who reported an absence due to respiratory illness. Adjusted for temperature and humidity lagged one and two days. Units are 10 ppb for O<sub>3</sub> and NO<sub>2</sub>, 10 µg/m<sup>3</sup> for PM<sub>10</sub>.

**Table 4.6.3.1 NUMBER OF ABSENCES (ALL CAUSE) IN RELATION TO LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Relative Risk per Unit Exposure</b>	<b>95% C.I.</b>	<b>P-Value</b>
PEAK O <sub>3</sub>	0.0097	(-0.0129, 0.0324)	0.40
PM <sub>10</sub>	0.0136	(-0.0211, 0.0483)	0.44
NO <sub>2</sub>	0.0319	(-0.0080, 0.0719)	0.12
Avg O <sub>3</sub>	-0.0067	(-0.0515, 0.0380)	0.77

Adjusted for community of residence, age, sex, race, asthma status, income, passive smoke, pets, and pests. Relative risk is per unit of exposure.

**Table 4.6.3.2 TOTAL DURATION OF ABSENCES (ALL CAUSE) IN RELATION TO LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Increase Per Unit Exposure</b>	<b>95% C.I.</b>	<b>P-Value</b>
PEAK O <sub>3</sub>	0.0294	(-0.0056, 0.0644)	0.10
PM <sub>10</sub>	0.0134	(-0.0400, 0.0668)	0.62
NO <sub>2</sub>	0.0239	(-0.0378, 0.0856)	0.45
Avg O <sub>3</sub>	0.0401	(-0.0292, 0.1095)	0.26

Adjusted for community of residence, age, sex, race, asthma status, income, smoke, weight, and houseplants. Relative risk is per unit of exposure.

**Table 4.6.3.3 NUMBER OF ABSENCES DUE TO RESPIRATORY ILLNESSES IN RELATION TO LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Relative Risk per Unit Exposure</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	0.0114	(0.0030, 0.0198)	0.01
PM <sub>10</sub>	0.0106	(-0.0017, 0.0229)	0.09
NO <sub>2</sub>	-0.0009	(-0.0157, 0.0140)	0.91
Avg O <sub>3</sub>	0.0154	(-0.0014, 0.0322)	0.07

Adjusted for community of residence, age, sex, race, asthma status, weight, mold or mildew, carpet, pets, income, Hispanicity, and vitamins. Relative risk is per unit of exposure.

**Table 4.6.3.4 TOTAL DURATION OF ABSENCES DUE TO RESPIRATORY ILLNESSES IN RELATION TO LIFETIME EXPOSURE TO AIR POLLUTANTS**

<b>Pollutant</b>	<b>Increase Per Unit Exposure</b>	<b>95% C.I.</b>	<b>P-value</b>
PEAK O <sub>3</sub>	0.0149	(0.0024, 0.0274)	0.02
PM <sub>10</sub>	0.0099	(-0.0085, 0.0284)	0.29
NO <sub>2</sub>	-0.0088	(-0.0307, 0.0132)	0.43
Avg O <sub>3</sub>	0.0317	(0.0066, 0.0569)	0.01

Adjusted for community of residence, age, sex, race, asthma status, weight, pests, vitamins, and carpet. Relative risk is per unit of exposure.

**TABLE 4.7.1 RELATIONSHIP BETWEEN FEV<sub>1</sub> AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

Pollutant	Coefficient	95% C.I.	P-value
Average O <sub>3</sub>	-0.24	(-1.30, 0.83)	0.66
PM <sub>10</sub>	-0.13	(-1.63, 1.37)	0.86
NO <sub>2</sub>	0.02	(-1.24, 1.28)	0.97
Acid	-4.59	(-17.32, 8.15)	0.48

FEV<sub>1</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb and acid (inorganic) in ppb.

Adjusted for age, sex, race, height, weight, hispanic origin, asthma, pets, and gas stove.

**TABLE 4.7.2 RELATIONSHIP BETWEEN FVC AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

Pollutant	Coefficient	95% C.I.	P-value
Average O <sub>3</sub>	-0.04	(-1.22, 1.14)	0.95
PM <sub>10</sub>	0.24	(-1.40, 1.87)	0.78
NO <sub>2</sub>	-0.59	(-1.97, 0.79)	0.40
Acid	-9.98	(-22.82, 3.90)	0.16

FVC is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb and acid (inorganic) in ppb.

Adjusted for age, sex, race, height, weight, hispanic origin, and gas stove.

**TABLE 4.7.3 RELATIONSHIP BETWEEN PEFR AND 1994 AMBIENT LEVELS OF AIR POLLUTANT**

Pollutant	Coefficient	95% C.I.	P-value
Ozone	0.46	(-3.03, 3.95)	0.80
PM <sub>10</sub>	-3.26	(-8.07, 1.56)	0.19
NO <sub>2</sub>	1.55	(-2.55, 5.65)	0.46
Acid	15.46	(-26.06, 56.96)	0.47

PEFR is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb and acid (inorganic) in ppb.

Adjusted for age, race, height, weight, pets, pests, exercise within 30 minutes of pulmonary function test, passive smoking.

**TABLE 4.7.4 RELATIONSHIP BETWEEN MMEF AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

Pollutant	Coefficient	95% C.I.	P-value
Ozone	-1.04	(-3.51, 1.44)	0.41
PM <sub>10</sub>	-2.46	(-5.90, 0.98)	0.16
NO <sub>2</sub>	-1.04	(-3.92, 1.85)	0.48
Acid	-9.94	(-47.47, 19.50)	0.51

MMEF is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb and acid (inorganic) in ppb.

Adjusted for age, race, sex, height, weight, asthma, hay fever, and passive smoking.

**TABLE 4.7.5 RELATIONSHIP BETWEEN ASTHMA AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
Average Ozone	1.00	(0.99, 1.01)	0.45
PM <sub>10</sub>	1.01	(0.99, 1.03)	0.28
NO <sub>2</sub>	1.01	(1.00, 1.02)	0.20
Acid	1.02	(0.86, 1.21)	0.82

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub>, in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for community, age, sex, race, height, weight, asthma, pets, hay fever, health insurance, pests, houseplants, and carpet. Odds ratios are per unit of exposure.

**TABLE 4.7.6 RELATIONSHIP BETWEEN WHEEZE AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

Pollutant	OR	95% C.I.	P-value
Average Ozone	1.00	(0.99, 1.01)	0.84
PM <sub>10</sub>	1.00	(0.97, 1.02)	0.75
NO <sub>2</sub>	1.00	(0.98, 1.01)	0.66
Acid	0.90	(0.72, 1.12)	0.34

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub>, in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for community, age, sex, race, and hay fever. Odds ratios are per unit of exposure.

**TABLE 4.7.7 RELATIONSHIP BETWEEN BRONCHITIS AND 1994 AMBIENT LEVELS OF AIR POLLUTANT**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Average Ozone	1.00	(1.00, 1.01)	0.47
PM <sub>10</sub>	1.00	(0.98, 1.01)	0.51
NO <sub>2</sub>	1.00	(0.99, 1.01)	0.53
Acid	0.99	(0.87, 1.14)	0.90

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for community, age, sex, race, hay fever, mold or mildew, and carpet. Odds ratios are per unit of exposure.

**TABLE 4.7.8 RELATIONSHIP BETWEEN COUGH AND 1994 AMBIENT LEVELS OF AIR POLLUTANTS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Average Ozone	1.00	(0.99, 1.01)	0.87
PM <sub>10</sub>	1.00	(0.98, 1.02)	0.69
NO <sub>2</sub>	0.99	(0.98, 1.01)	0.48
Acid	0.97	(0.79, 1.19)	0.76

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub> in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for community, age, sex, race, hay fever, vitamins, height, income, and mold or mildew. Odds ratios are per unit of exposure.



**TABLE 4.7.9 RELATIONSHIP BETWEEN PNEUMONIA AND 1994 AMBIENT LEVELS OF AIR POLLUTANT**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Average Ozone	1.00	(0.99, 1.01)	0.88
PM <sub>10</sub>	1.00	(0.97, 1.03)	0.77
NO <sub>2</sub>	1.00	(0.98, 1.03)	0.75
Acid	1.03	(0.77, 1.37)	0.84

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub>, in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for health insurance. Odds ratios are per unit of exposure.

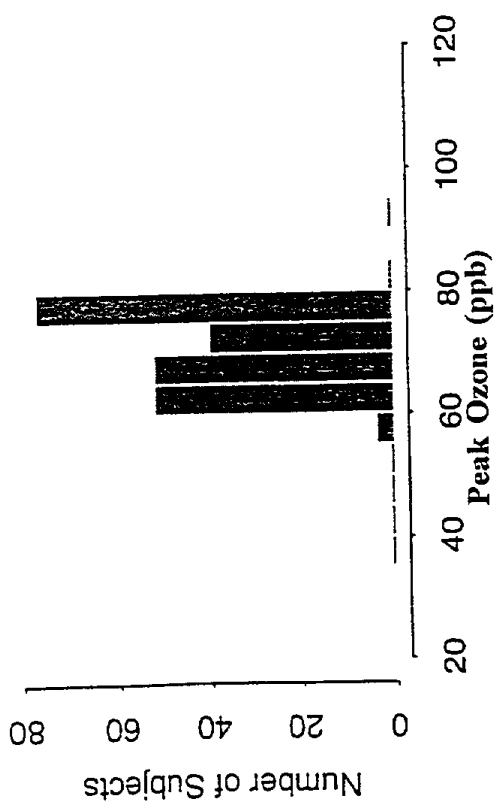
**TABLE 4.7.10 RELATIONSHIP BETWEEN OTHER CHEST ILLNESS AND 1994 AMBIENT LEVELS**

<b>Pollutant</b>	<b>OR</b>	<b>95% C.I.</b>	<b>P-value</b>
Average Ozone	1.00	(0.99, 1.01)	0.67
PM <sub>10</sub>	1.00	(0.98, 1.02)	0.67
NO <sub>2</sub>	1.00	(0.98, 1.02)	0.99
Acid	0.95	(0.77, 1.17)	0.62

O<sub>3</sub> is measured in ml, O<sub>3</sub> in ppb, PM<sub>10</sub>, in µg/m<sup>3</sup>, NO<sub>2</sub> in ppb. Inorganic acid is measured in standard units using the mean standard deviation for the 12 communities. The unit is the difference in mean between the community of interest and the mean of all 12 communities expressed in standard deviation units. Adjusted for community, age, sex, race, hay fever, vitamins, and mold or mildew. Odds ratios are per unit of exposure.

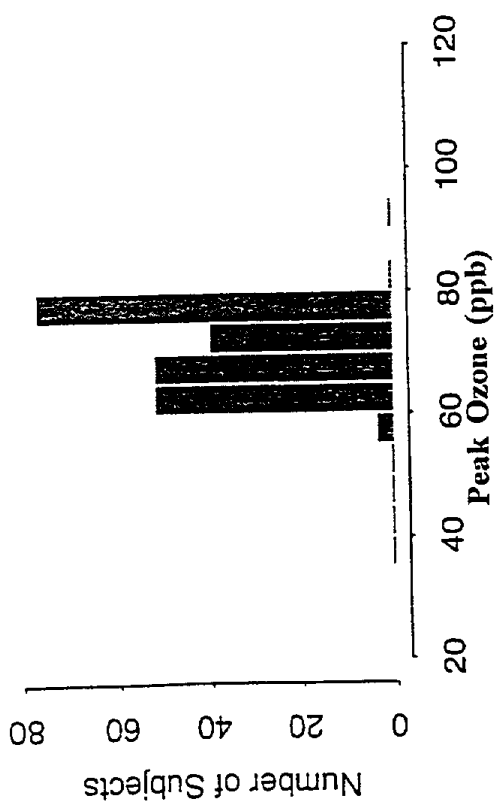
# Alpine

ESTIMATED LIFETIME EXPOSURE (1994)



# Lake Arrowhead

ESTIMATED LIFETIME EXPOSURE (1994)



# Lancaster

# Lake Elsinore

ESTIMATED LIFETIME EXPOSURE (1994)

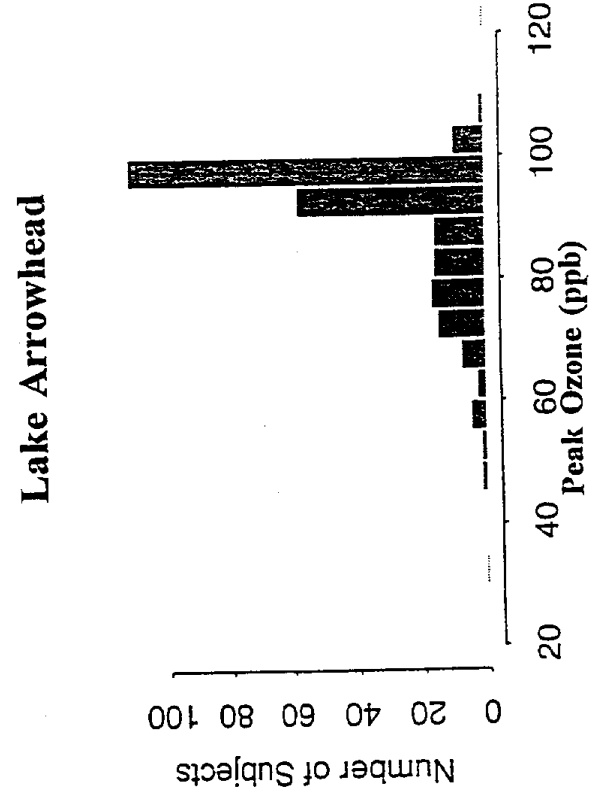


FIGURE 4.1.3.1

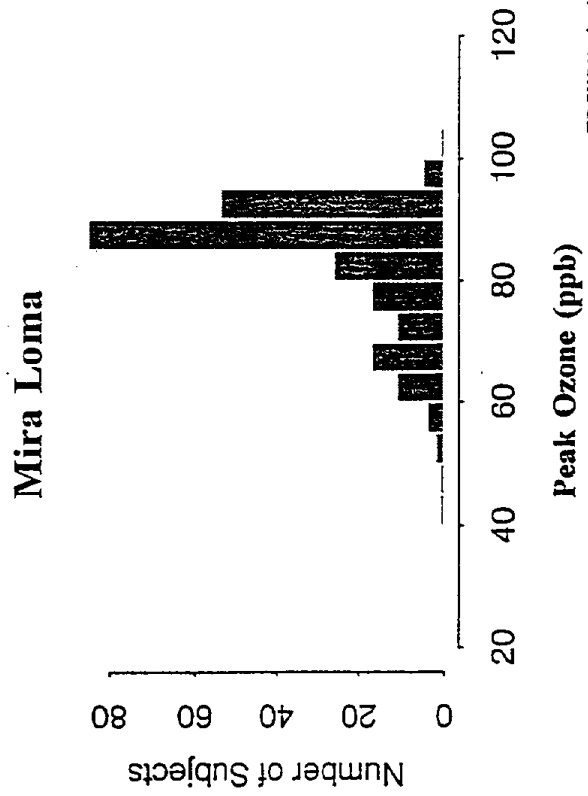
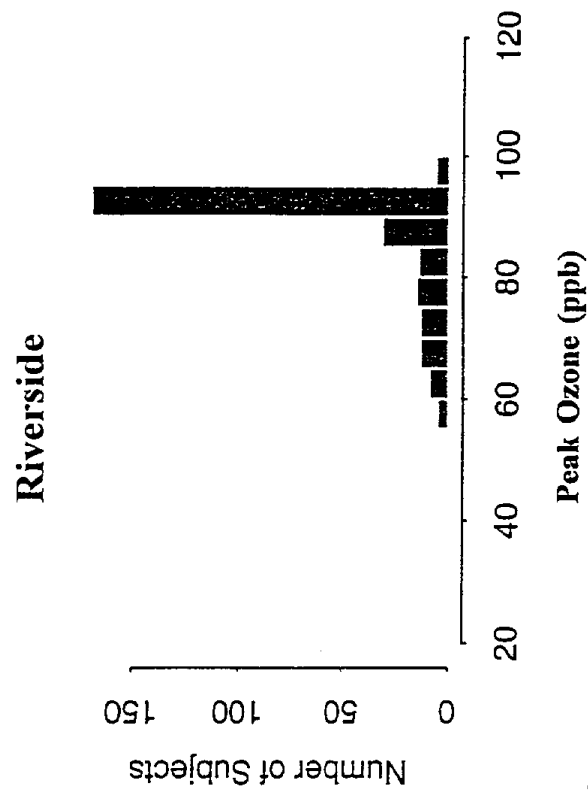
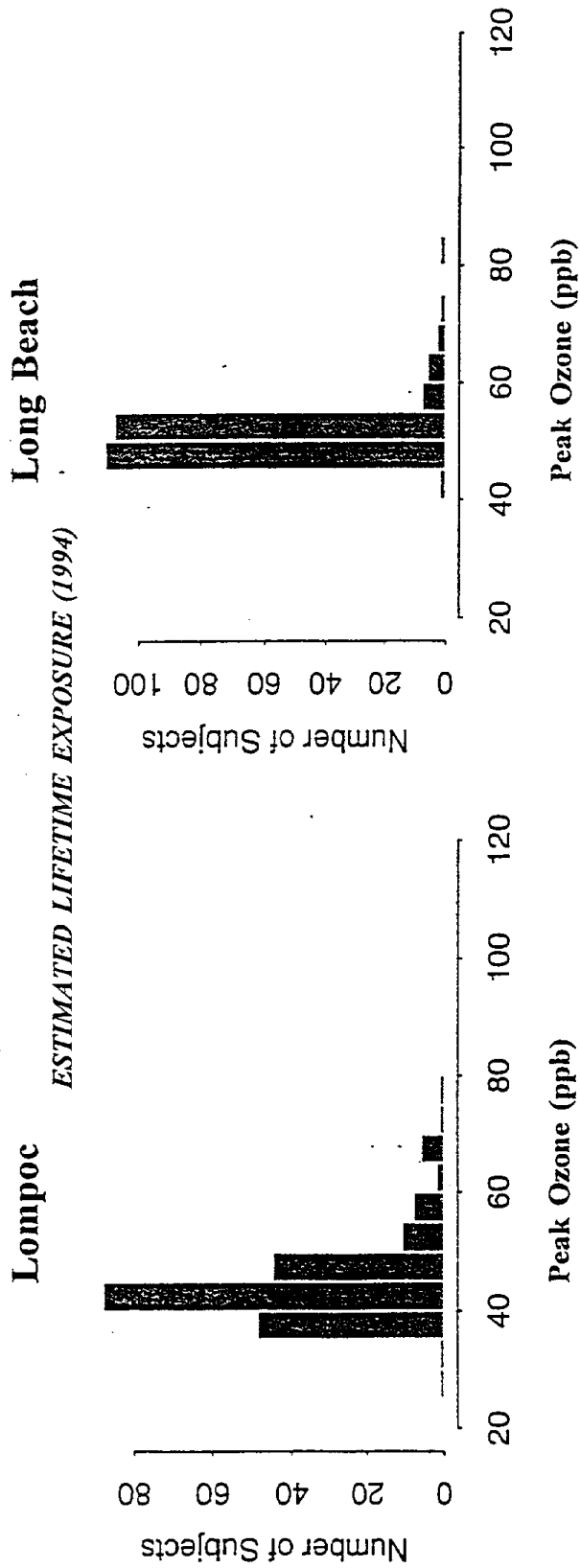


FIGURE 4.1.3.2

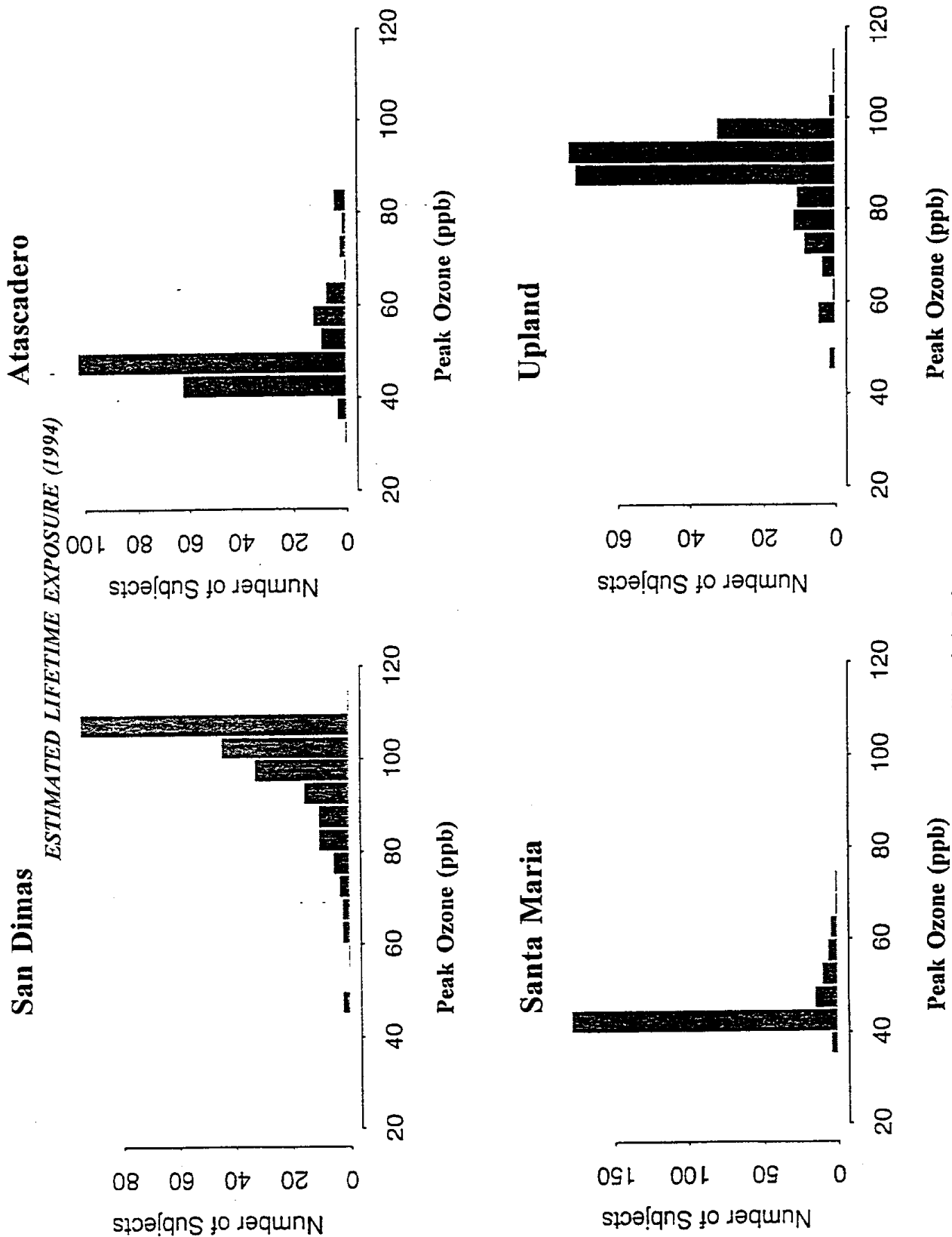
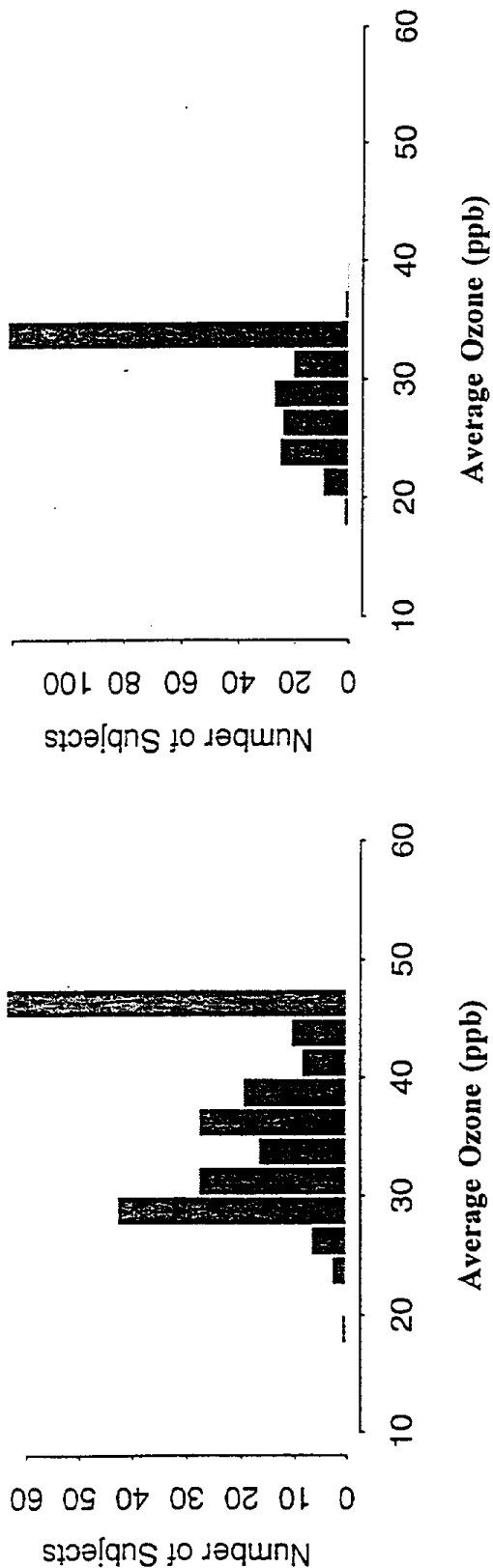


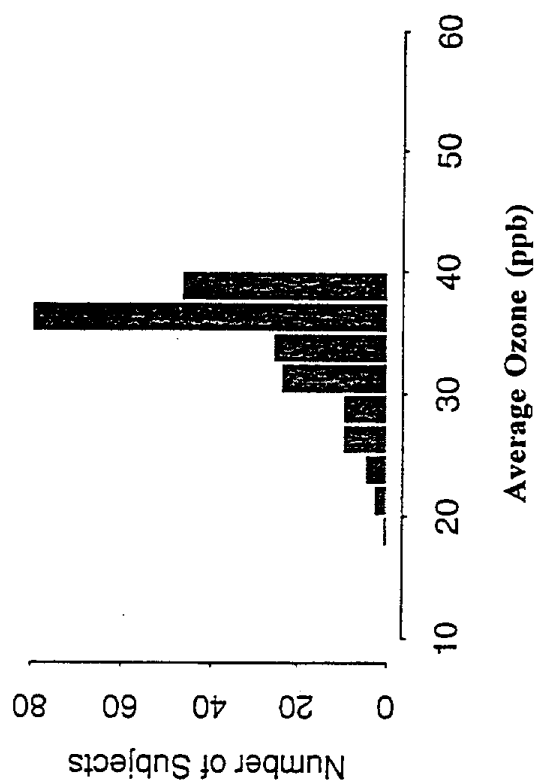
FIGURE 4.1.3.3

**Alpine** **Lake Elsinore**  
*ESTIMATED LIFETIME EXPOSURE (1994)*



58-4

**Lancaster**



**Lake Arrowhead**

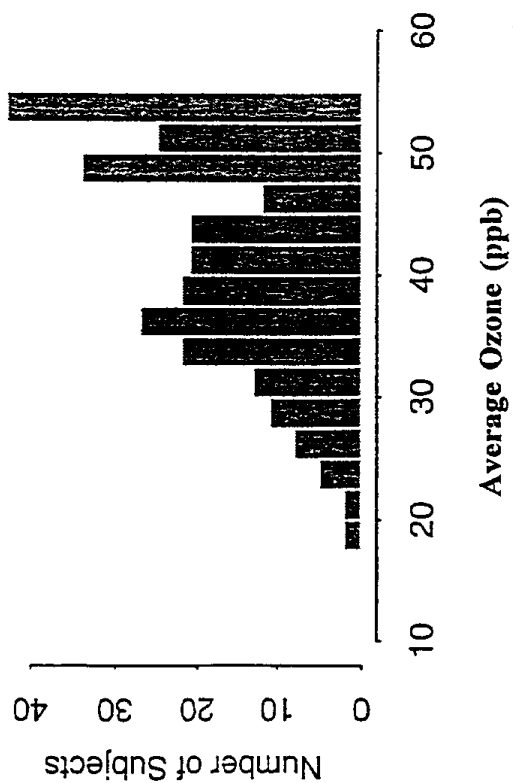
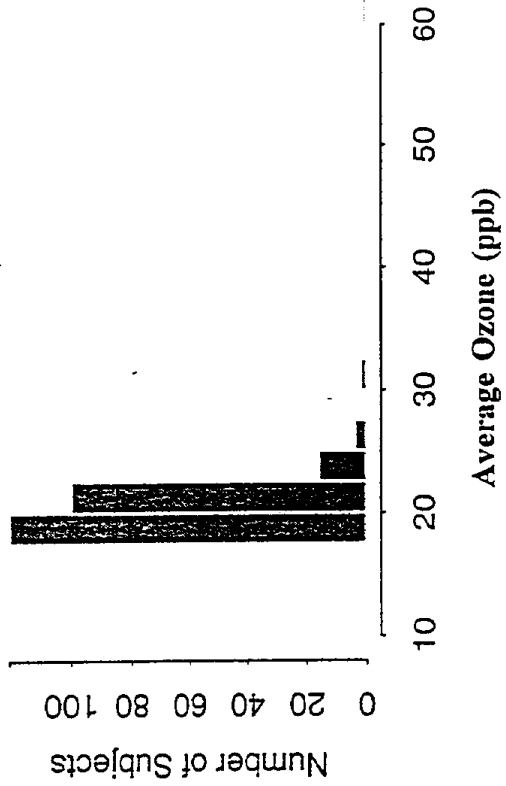
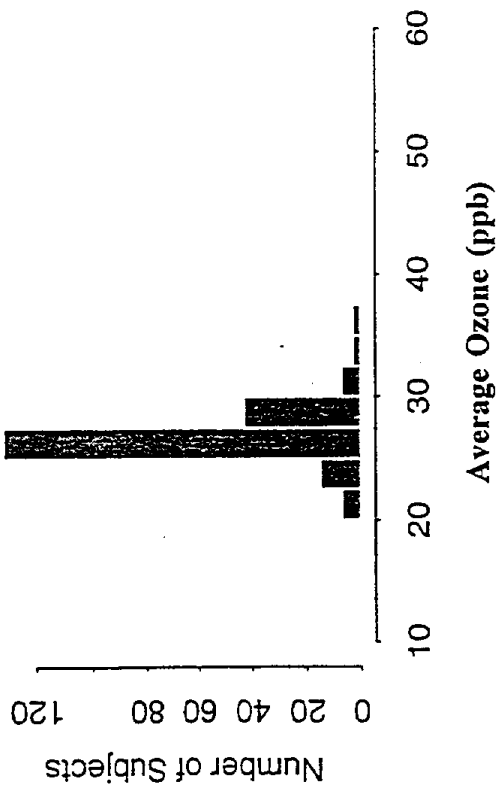


FIGURE 4.1.3.4

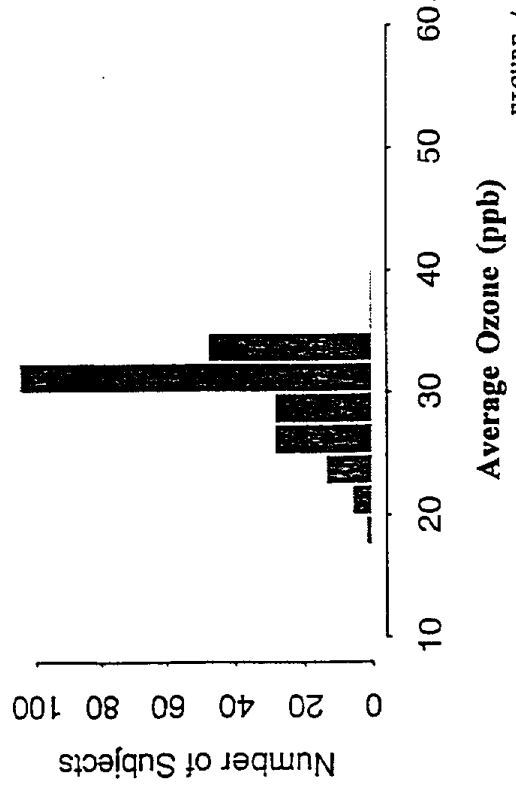
# Lompoc

ESTIMATED LIFETIME EXPOSURE (1994)

# Long Beach



# Mira Loma



# Riverside

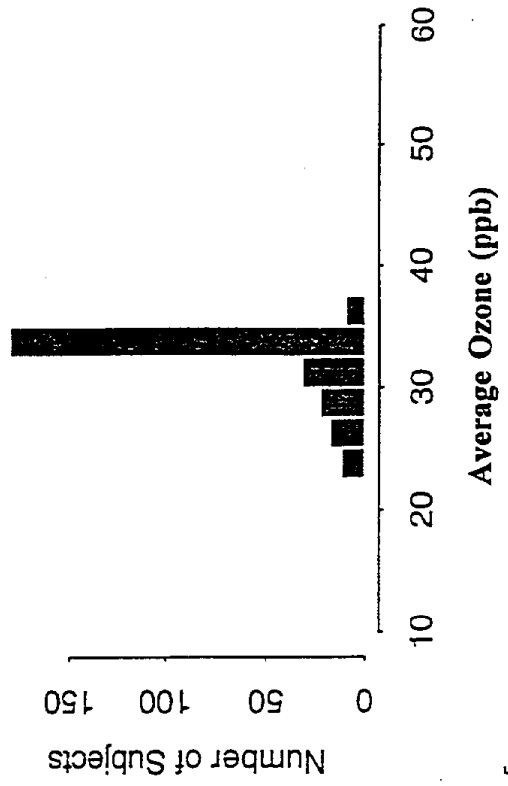
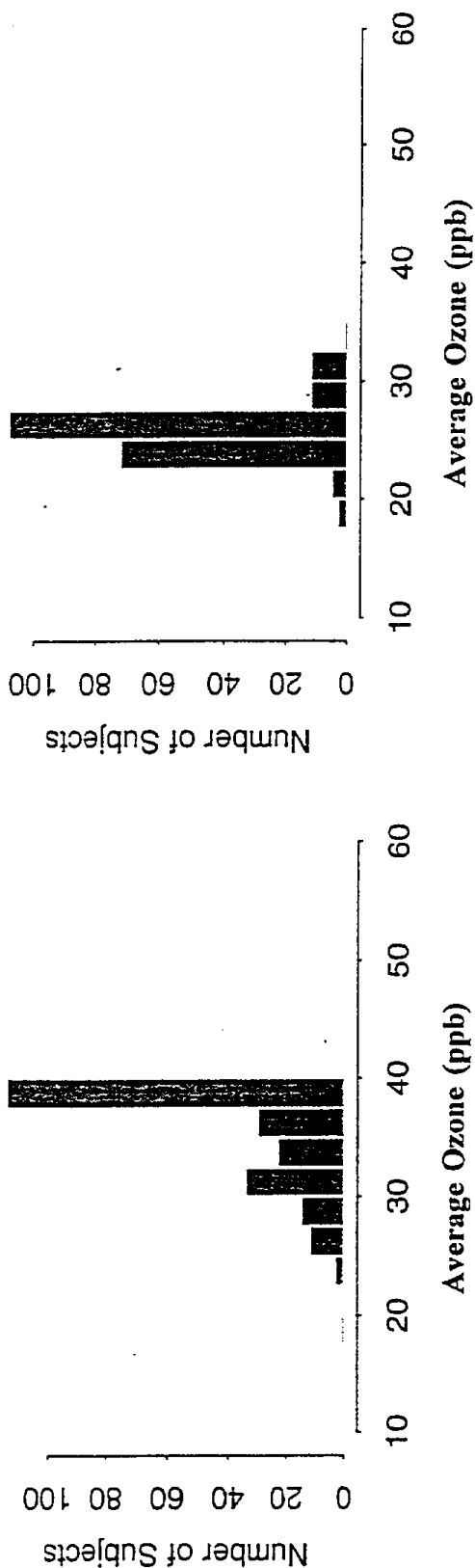


FIGURE 4.1.3.5

# San Dimas                      Atascadero

## ESTIMATED LIFETIME EXPOSURE (1994)



4-87

# Upland

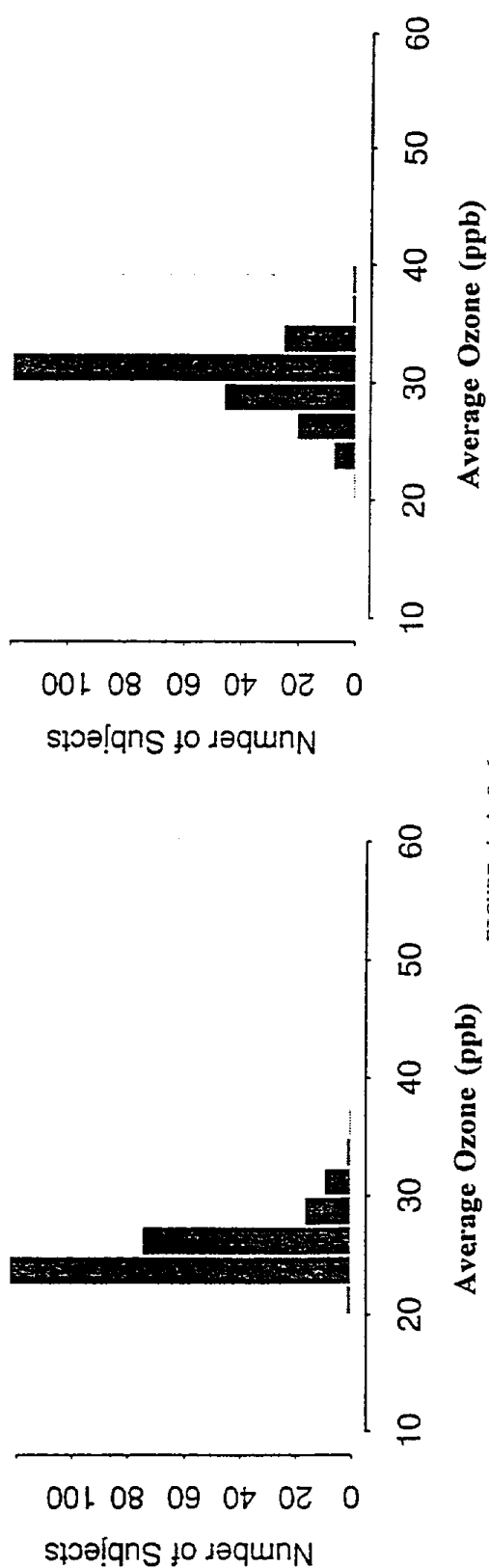


FIGURE 4.1.3.6

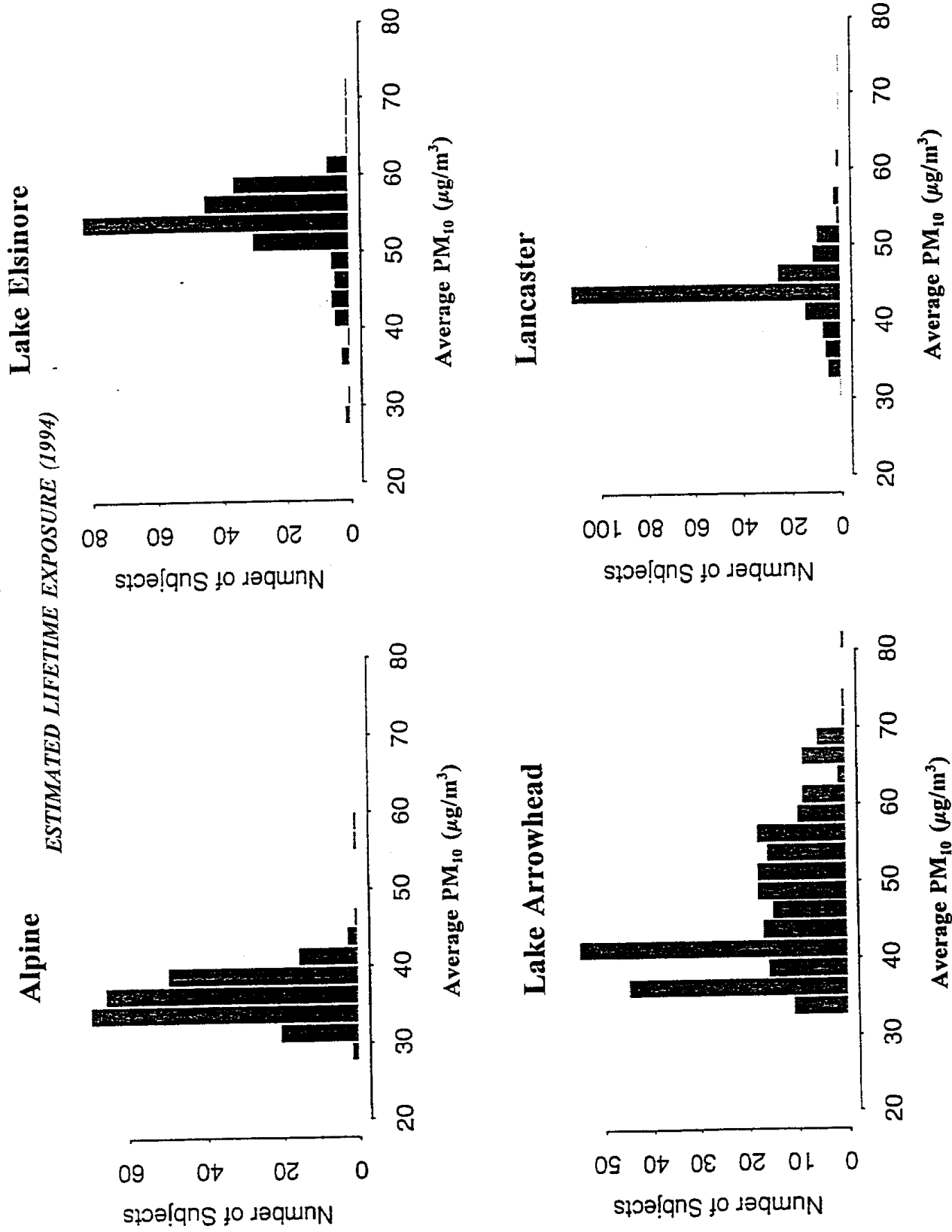
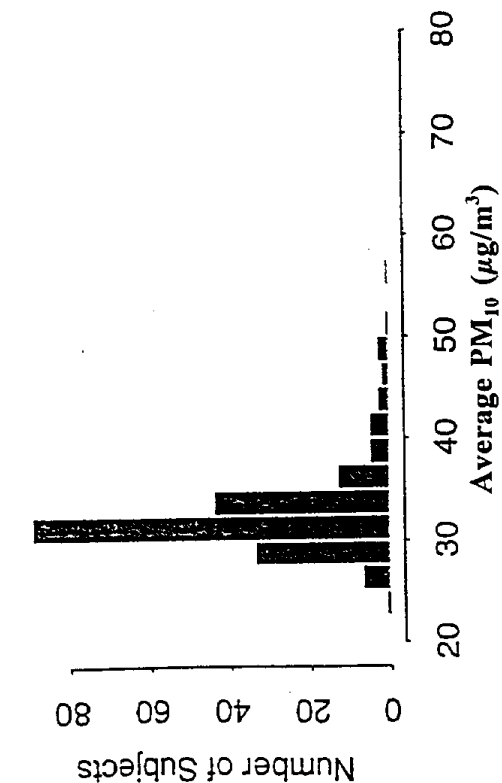


FIGURE 4.1.3.7

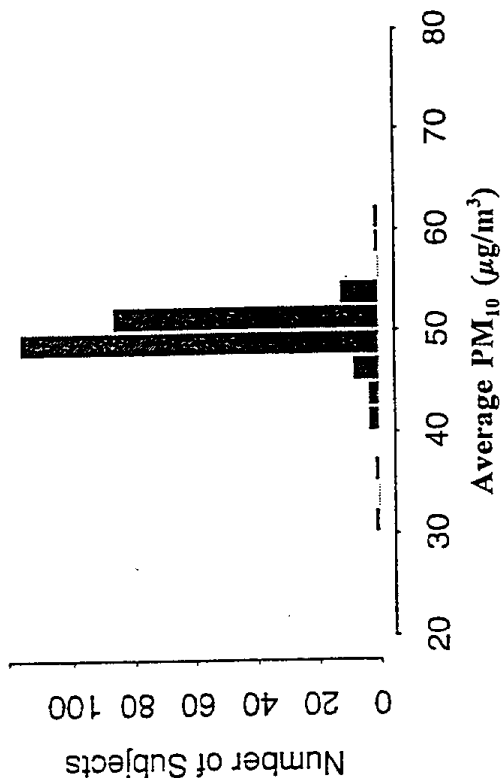


# Lompoc

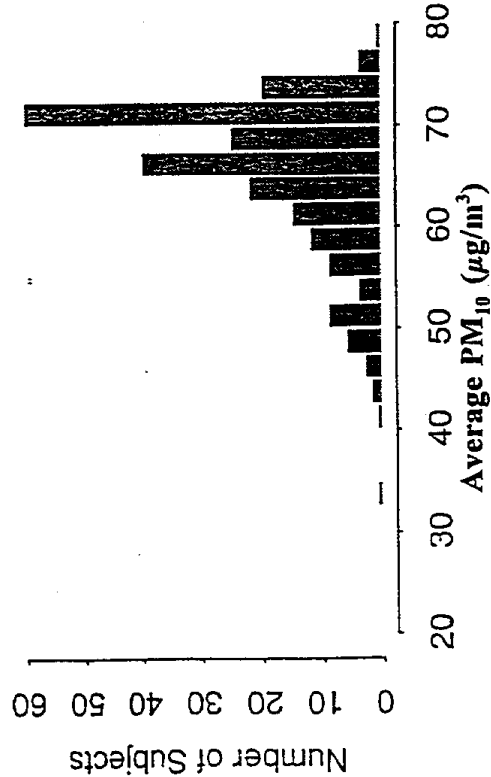
ESTIMATED LIFETIME EXPOSURE (1994)



# Long Beach



# Mira Loma



# Riverside

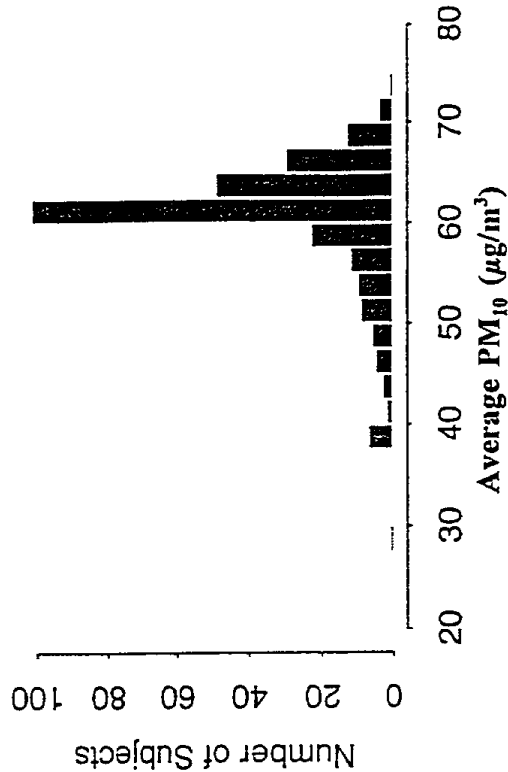
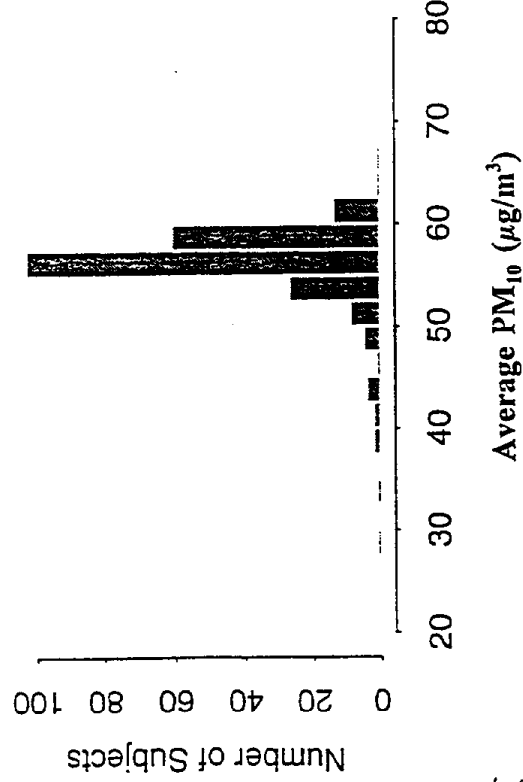


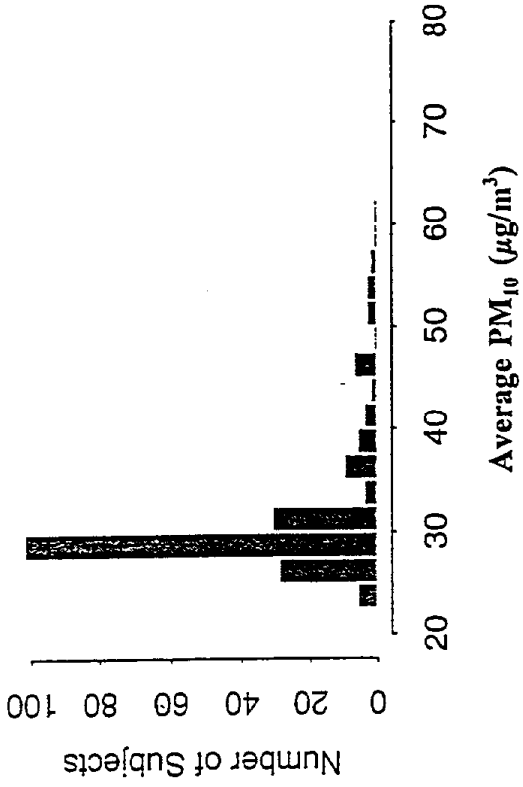
FIGURE 4.1.3.8

# San Dimas

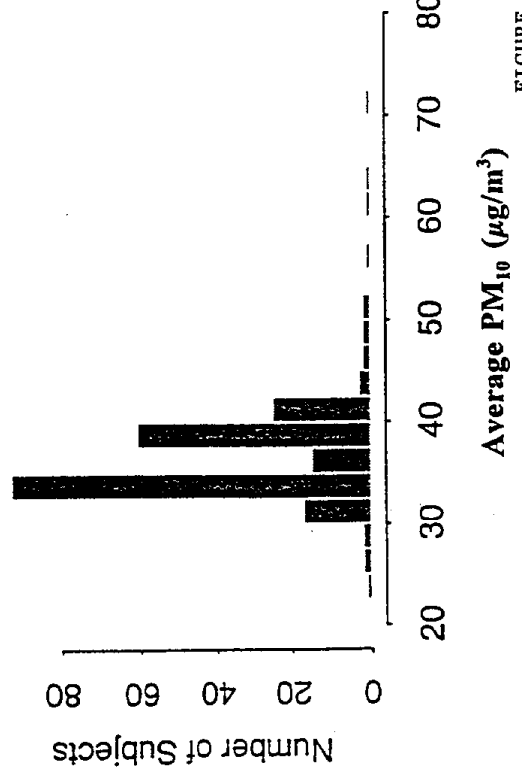
ESTIMATED LIFETIME EXPOSURE (1994)



# Atascadero



# Santa Maria



# Upland

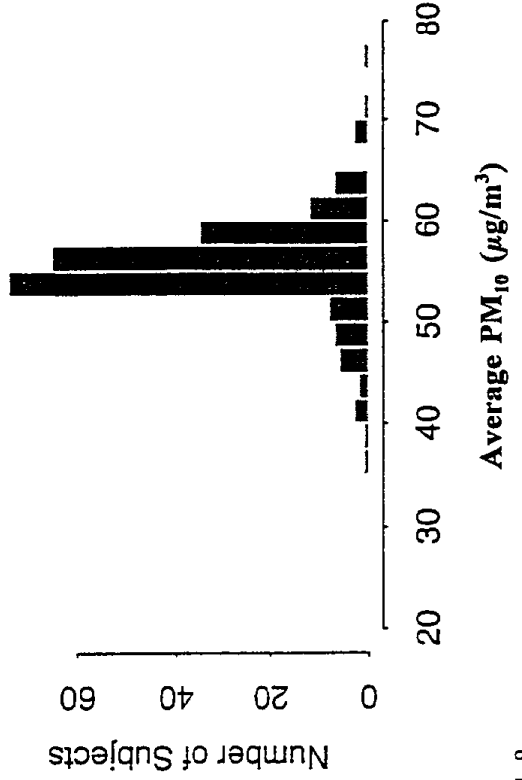
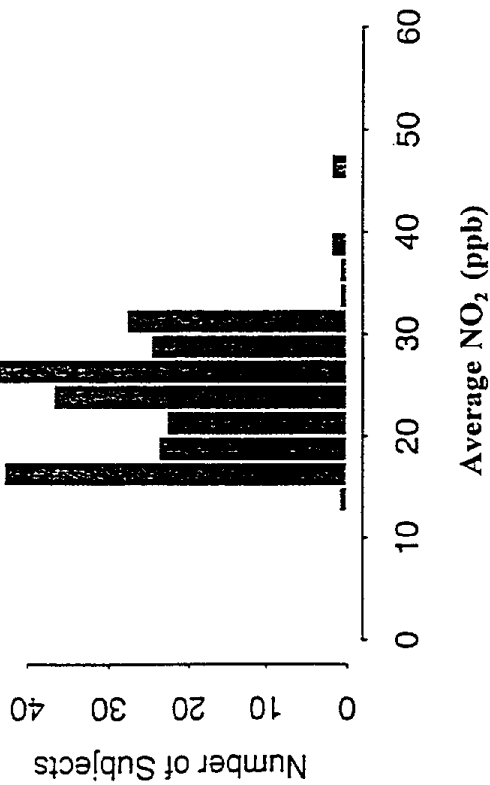


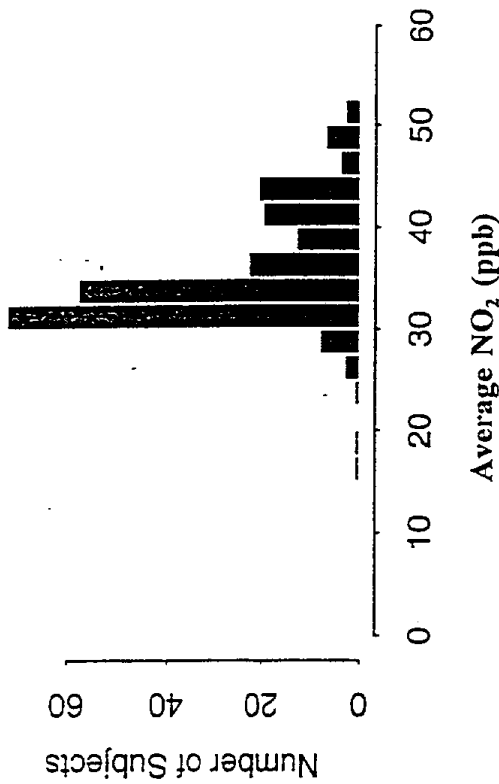
FIGURE 4.1.3.9

# Alpine

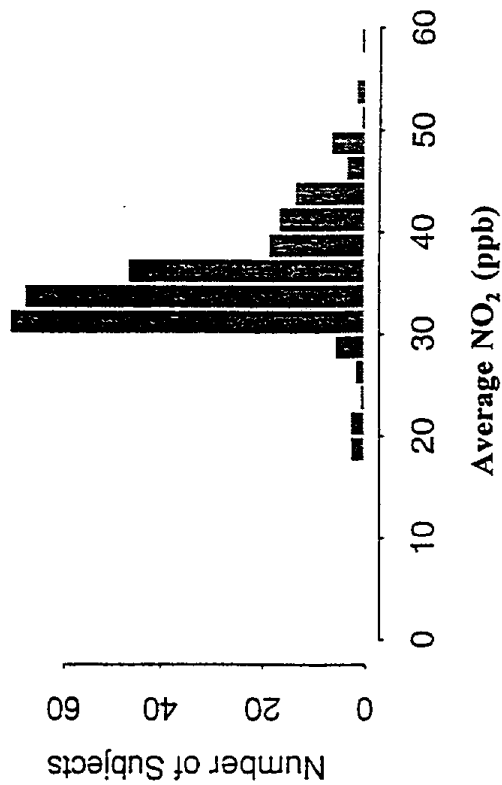
ESTIMATED LIFETIME EXPOSURE (1994)



# Lake Elsinore



# Lake Arrowhead



# Lancaster

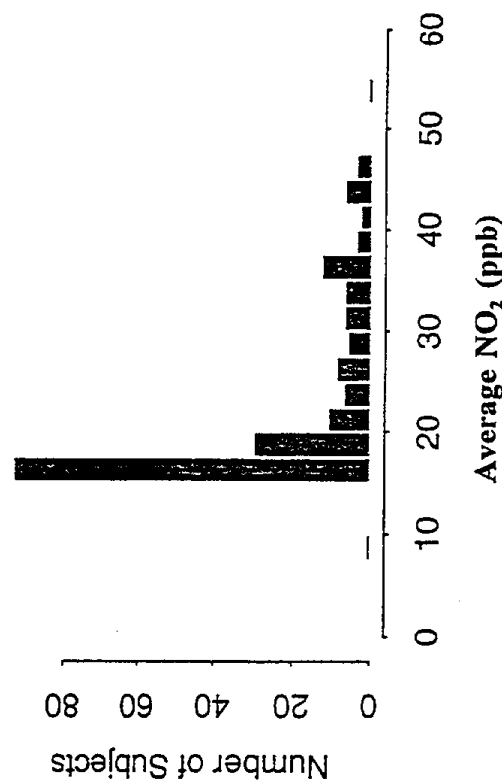
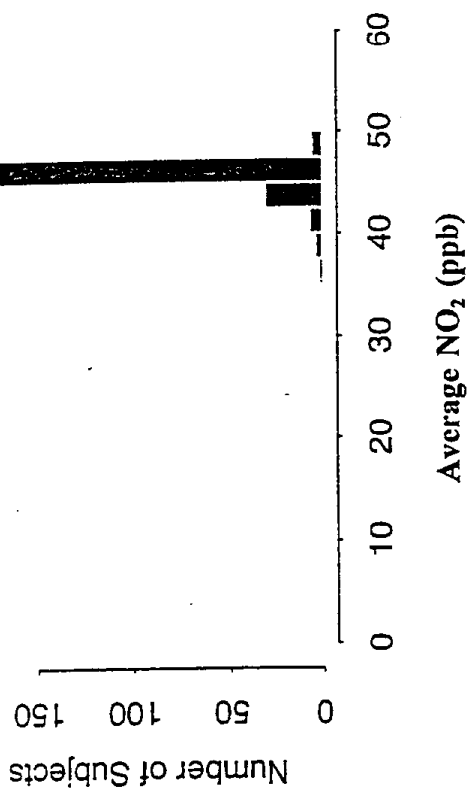
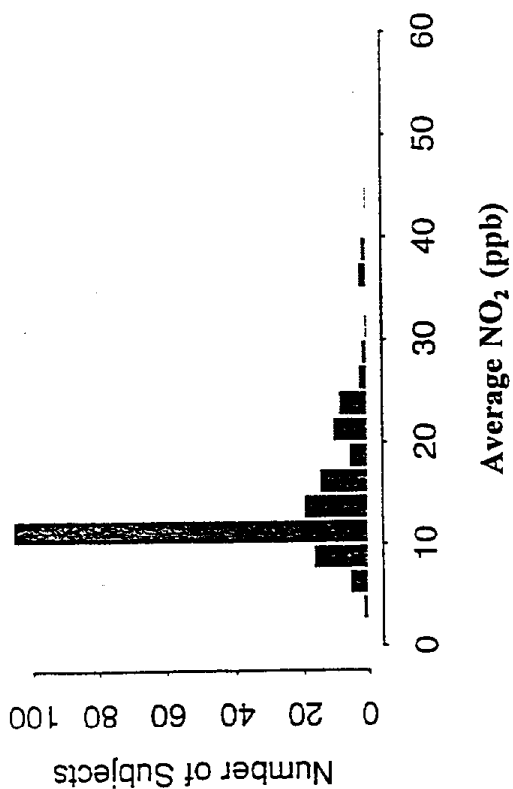


FIGURE 4.1.3.10

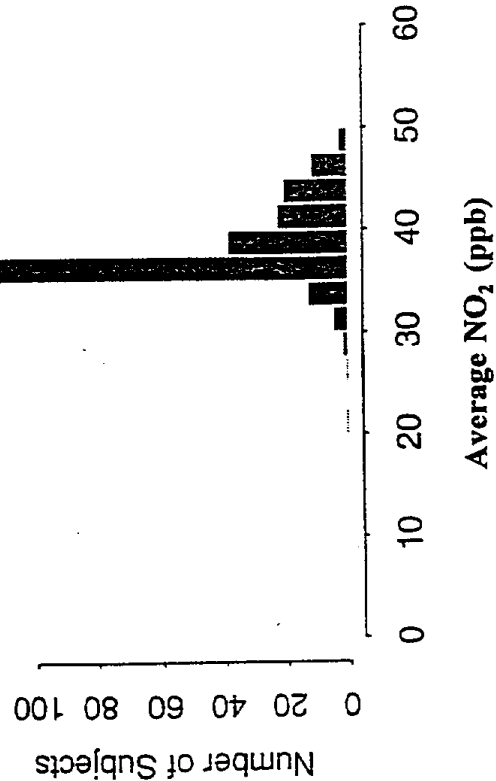
# Lompoc

ESTIMATED LIFETIME EXPOSURE (1994)

# Long Beach



# Mira Loma



# Riverside

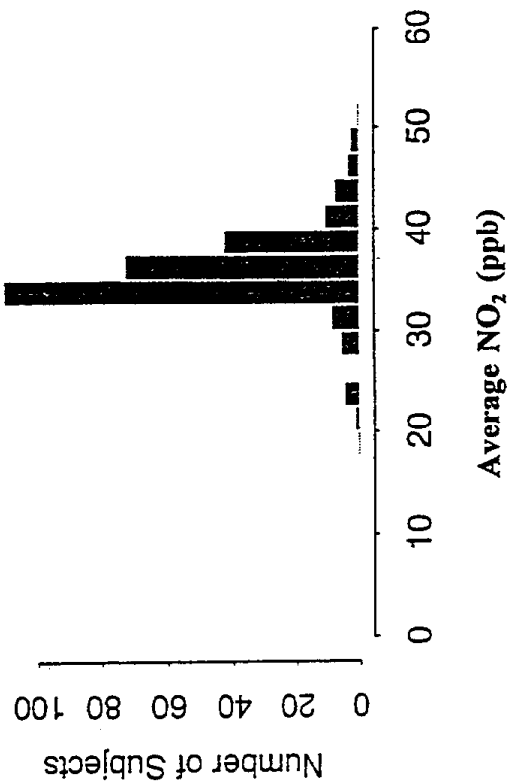
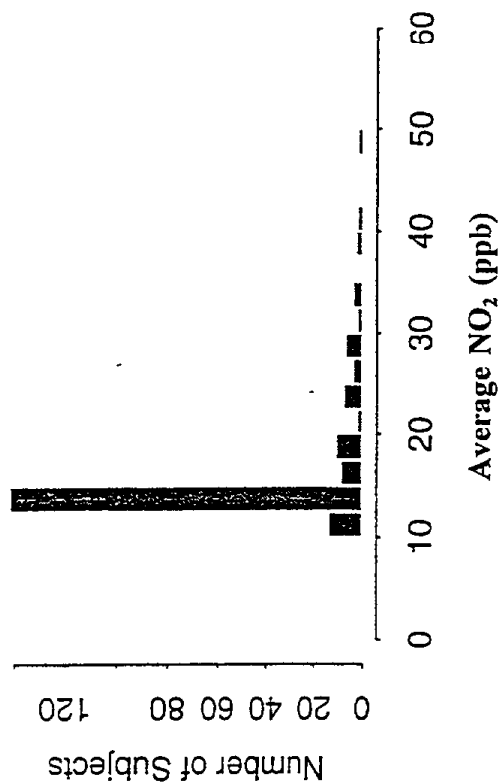
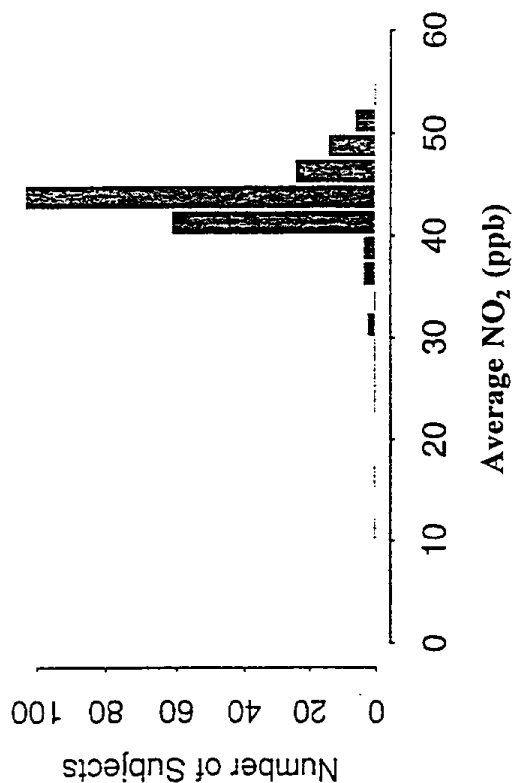
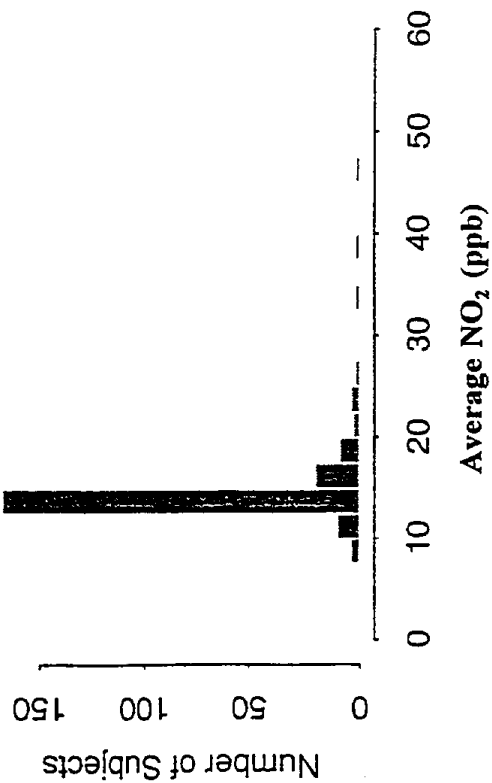


FIGURE 4.1.3.11

# **San Dimas** *ESTIMATED LIFETIME EXPOSURE (1994)* **Atascadero**



## **Santa Maria**



## **Upland**

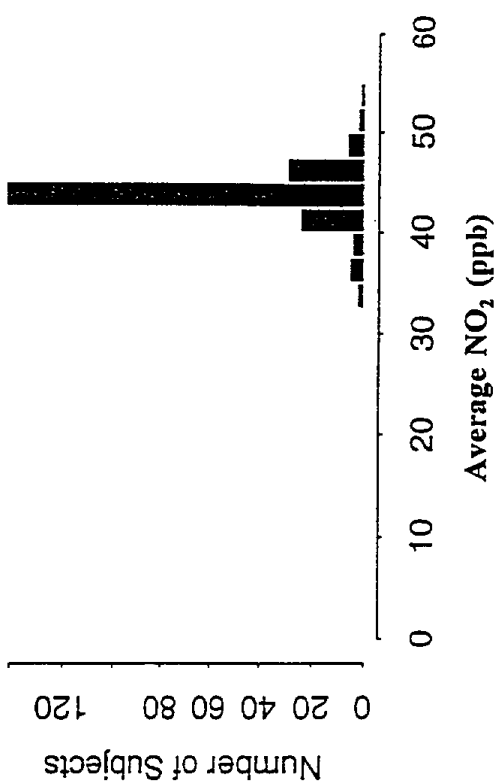


FIGURE 4.1.3.12



## **5. REPORT ON QUALITY ASSURANCE AND QUALITY CONTROL**

The quality assurance and quality control (QA/QC) activities were organized to follow the major operational components of the study. These components include exposure assessment, lung function measurements, questionnaire surveys, and data management. Exposure assessment operations were further subdivided into operation of community monitoring stations, indoor/outdoor sampling of ozone at schools, and laboratory operations. A Quality Assurance Plan was developed that describes the measures taken to control and to assess data quality for all aspects of the study (Peters et al., 1994). The QA Plan addresses the following elements for each component of the study:

- overview of QA for that component
- study design and method descriptions
- detailed standard operating procedures
- project management and reporting structure
- personnel qualifications and training
- maintenance of written records
- data processing/data management
- data validation and data quality assessments
- external quality assurance

The responsibilities for QA/QC were distributed among members of the ARB/USC Epidemiologic Study Team (the Study Team). Collins Consulting & Computing (CCC) provided overall coordination of QA/QC activities for the study, performed independent audits of exposure assessment monitoring conducted by the Sonoma Technology Inc. (STI), and performed independent audits of chemistry laboratory operations conducted by the Los Amigos Research and Education Institute (LAREI) in support of ambient and microenvironmental air quality monitoring. Another branch of LAREI provided independent QA for the pulmonary function measurements conducted by USC, including independent audits of operations and assessments of routine QC data. Results of the QA activities were reported in a series of regular reports to the Principal Investigator.

This section of the report summarizes the status and results of the Phase II quality assurance activities. The status issues include the development of the QA Plan and Standard Operating Procedures (SOPs), the development and acceptance testing of new monitoring methods, the implementation and reporting of regular QC checks, and the performance of external audits. Quantitative results of developmental testing, QC checks, and external audits are summarized where applicable. Detailed information on the quality assurance program are provided by Peters et al., (1994).

## **5.1 EXPOSURE ASSESSMENTS**

### **5.1.1 Continuous Air Quality Analyzers**

Continuous ambient air quality analyzers were operated in the twelve communities included in the study. Five of the air quality monitoring stations were new sites that were installed and initially operated by STI. Operation of the five new sites was transferred from STI to ARB in October 1994. The other seven stations were existing sites that were operated by local Air Quality Management and Air Pollution Control Districts, and by the Air Resources Board. Operations by state and local agencies are ongoing and well established operations conducted according to applicable EPA guidelines for routine ambient monitoring. The agency operated stations were augmented by addition of monitoring equipment where necessary to achieve a uniform collection of monitored parameters:  $O_3$ ,  $NO_2$ , and  $PM_{10}$ . The monitors added to existing sites passed acceptance testing conducted by the ARB before being installed in the monitoring stations. Responsibility for operation of the added monitors, including calibration, quality control, auditing, and data reporting, generally lies with the agency responsible for the monitoring station. The results of 1994 audits conducted by the government agencies and CCC are summarized below.

STI setup and initially operated five newly established monitoring stations for the study. In addition, STI was responsible for operation of the monitors added to the existing Santa Maria and Alpine stations. All of the newly installed monitors passed acceptance testing conducted by the ARB before being installed. All aspects of operations for the continuous gas analyzers, including site selection, equipment set up, calibration, and quality control, followed applicable EPA guidelines for ambient monitoring (EPA, 1977, as revised through 1992) to the extent practical. The Tapered Element Oscillating Microbalance (TEOM) continuous  $PM_{10}$  monitors were installed and operated in accordance with the manufacturer's recommendations.

#### **QA Plan and SOPs for Continuous Analyzers**

Documentation for continuous analyzer operations is provided in the Continuous Monitoring QA Plan (Wright et al., 1994) and Peters et al., (1994). In addition, Lurmann et al., (1994) provides an overview of site selection, instrumentation, operation, station documentation, data management and data validation. The QA Plan addresses each of the important QA elements and provides site and sampler placement identification reports, instrument specifications, and SOPs for calibration, precision checks, and operation.

#### **QC, Validation, and Data Results**

Daily zero/span checks, data capture, and bi-weekly precision checks were regularly reviewed by the field manager. These data in combination with the field and instrument



log books were used to validate or invalidate raw data on entry into the data base system at the STI field office. On entry into the data base system at the field office, quantitative checks for outlier values and outlier rate of change were automatically performed and anomalies flagged. The preliminary data were sent to the data management staff at STI's central office. The data were double-checked and either returned to the field office or approved and entered into the database system as preliminary data. Preliminary data were reported monthly to the program manger and a copy sent to the field manager. On approval by the field manager and the program manger, the monthly data were designated as final data. The final data were reported to the overall data manger at USC after the first six months and then quarterly thereafter. The data reports included accuracy and precision data.

Quality assured data from continuous monitoring instruments operated by the South Coast Air Quality Management District (SCAQMD), San Diego Air Pollution Control District (SDAPCD), and the San Luis Obispo County Air Pollution Control District (SLOAPCD) were obtained quarterly, reviewed by STI, and incorporated into the project data archive.

### **External Audits**

The audit frequency for the agency operated sites were generally at least once per year. The audits were performed by independent QA branches of the monitoring agencies, and/or by separate agencies. Results for audits of agency sites are summarized in Table 5-1 along with the results for audits of the STI-operated sites.

Audit frequency for the new monitoring sites were approximately once per six months. A system and performance audit of the continuous monitoring equipment operated by STI at the new monitoring sites was conducted by CCC during February 1994, for equipment operational at that time (Collins, 1994). Station measurements for the performance audits were obtained from the station data logger and corrected using instrument calibration factors supplied by the station operator. In addition, the ARB QA group performed an audit of all twelve sites in August 1994.

The CCC audit procedures were based on EPA guidelines (EPA, 1977, as revised through 1992) with the following two exceptions. First, audit test gases were introduced to the sampling system at a point 3 to 4 meters downstream of the inlet filter on the sampling line instead of upstream of the filter. Second, the transfer standard for the ozone audit test gas was a stable, calibrated ozone generator. This generator was calibrated against primary standard and transfer standard photometers, but a transfer standard photometer was not used in the field.

The first difference from EPA guidelines was due to practical constraints on the equipment setup. This difference does not affect the ability of the audit to characterize the new analyzers' calibration and linearity, but does mean that sample transfer from the

ambient atmosphere to the analyzer through the teflon filter and sample line was not tested during the audit.

The use of a stable calibrated ozone generator as the transfer standard was based on convenience considerations and supported by an EPA verification of the calibration for the generator. However, this system underwent a shift in calibration between EPA verification and use in the field. Future audits should incorporate a transfer standard photometer in the field during audit procedures.

Use of the EPA certified generator calibration values resulted in a consistent bias between the calibrated generator transfer standard and the new ozone analyzers. The generator was then "recalibrated" against an ARB transfer standard photometer, immediately after conclusion of the field audits. This ARB calibration of the generator demonstrated a shift in generator performance with respect to the earlier EPA certified calibration values. The ARB calibration values were then applied retroactively to the field audit data to determine audit input values. Use of these ARB calibrated audit values reduced the bias between the generator and the new ozone analyzers to insignificant levels, as shown in Table 5-1.

Performance audits conducted by CCC for the TEOMs consisted of observing all front panel readouts, checking flow rates with a calibrated orifice at the PM<sub>10</sub> sampling head and, for some sites, at the inlet to the TEOM flow control module. The orifice and pressure gage employed were calibrated against a certified wet test meter. A calibrated orifice was chosen as the audit device in preference to a mass flow meter for two reasons: (1) the orifice is a simple fundamental physical system which is not subject to electronic drifts, temperature drifts, dirt, or clogging, and (2) the orifice introduces a very low pressure drop, and thus modifies the flow rate only very slightly (a feature essential for Two-week Sampler (TWS) audits conducted during the same audit visit). However, the orifice design employed was influenced by variations in the ambient wind speeds. Conditions during the audits were often rainy and windy, which made stable readings difficult to obtain. Future audits should use an orifice set-up less susceptible to ambient winds. The ARB QA group used a mass-flow meter to audit the TEOM flows.

### **System Audits**

The results of the system audits can be summarized as follows. The new monitoring stations were spacious and temperature-controlled. The stations and sample probes were located in conformance with EPA guidelines and representative of the surrounding area. All equipment was functional, well organized, and readily accessible. Automated calibration equipment generated daily precision checks. Span gas cylinder certifications were current. The site technician was very knowledgeable and kept detailed instrument and station logs. Tools and supplies were carried with the site technician. Operational status and daily precision check results were monitored daily by telemetry. Stations were visited at least every two weeks, or within one day if telemetry

indicated a problem. Instrument manuals were kept on site; SOPs for calibration and precision checks were in possession of the site technician.

### **Nitrogen Dioxide and Ozone Performance Audit Results**

Nitrogen dioxide and ozone performance audit results are summarized in terms of average percent difference in Table 5-1. EPA suggests that exceeding  $\pm 15$  percent for the slope or exceeding  $\pm 3$  percent of full-scale (i.e., generally  $\pm 15$  or  $\pm 30$  ppb) for the intercept of a 4-point-plus-zero least squares regression is unsatisfactory. A slope within  $\pm 5$  percent is considered excellent. The results for average percent difference are all consistent with the EPA suggested criteria. The linear regression slopes at all locations were within  $\pm 5$  percent, in the excellent range. Zero intercepts were within EPA criteria tolerance, but negative intercepts sometimes contributed to lower the average percent difference. The input concentrations used were roughly 100, 200, and 400 ppb, appropriate for a 500 ppb full-scale range. However, the instruments were operated on the 1000 ppb full-scale range. The influence of zero offsets on the average percent difference would have been smaller had higher audit concentrations been used.

### **TEOM Performance Audit Results**

TEOM performance audit results are shown in Table 5-2. For  $PM_{10}$  measurements by dichotomous samplers, EPA recommends "differences exceeding  $\pm 7$  percent require sampler recalibration. Differences exceeding  $\pm 10$  percent may result in invalidation of all data subsequent to the last calibration check or valid flow check" (EPA, 1977, revised 1990). The agreement between the audit and station values for main sampler flow rates meet these criteria, while the agreement between the audit and nominal values for total flow rate sometimes slightly exceed the criteria. We believe that the performance shown in Table 5-2 demonstrates adequate agreement for between station and audit values for the conditions of this audit. The reasons for this belief are twofold. First, the audit results were only slightly above the  $\pm 10$  percent criteria and discrepancies in these outdoor measurements could be due to the variations in ambient wind speeds. Some additional audit measurements were made indoors (out of the wind and rain) and agreed within  $\pm 10$  percent. Second, the TEOM  $PM_{10}$  mass values are calculated only from the main flow rate in the TEOM, for which the audit and station values were in good agreement. The TEOM total flow rate (main plus auxiliary) only affects the  $PM_{10}$  cut-point size. Note that a larger flow rate than the nominal rate will result in a slightly lower cut-point than  $10\mu m$ .

#### **5.1.2 Two-Week Sampler**

The Two-Week Samplers (TWS) are designed to provide 14-day integrated measurements of the following ambient pollutants:  $PM_{2.5}$  particle mass,  $PM_{2.5}$  nitrate,  $PM_{2.5}$  sulfate,  $PM_{2.5}$  ammonium,  $PM_{2.5}$  chloride, nitric acid, hydrochloric acid, formic acid, and acetic acid. TWS were installed at the twelve community monitoring

stations and operated by STI through December 1994. TWS field operations were transferred to ARB after December 1994. Los Amigos Research and Education Institute (LAREI) was responsible for filter and denuder preparation, sampler holder assembly and disassembly, chemical analysis of the samplers, and reporting laboratory data to STI. STI was responsible for combining laboratory data with field data, calculating final concentrations, and reporting final data to the overall data manager.

### **Sampler Development and Testing**

The TWS was a new sampler that was developed specifically for use in the Epidemiologic Study. It was designed and tested following a well-designed protocol. The sampler underwent extensive laboratory and field testing during the development and acceptance process. Detailed explanations of the need for the new sampler, the rationale guiding its design, laboratory test results, field test results, and final precision and accuracy estimates are provided by Lurmann et al., (1994). Additional precision data is collected through on-going deployment of collocated samplers at selected locations (see Table 3-10). Some key features of TWS performance are briefly discussed below.

The accuracy of the TWS varies with species. The ambient field evaluation showed that TWS fine particle sulfate and ammonium concentrations were 10 and 7 percent lower than those measured by a modified Southern California Air Quality Study (SCAQS) sampler (the best available reference method in 1993), respectively. This bias is most likely due to electrostatic losses of fine particles in the Teflon impactor and sampling system. A Teflon system was chosen in order to avoid loss of nitric acid to the sampler walls. Efforts to eliminate the electrostatic losses of fine particles in the Teflon inlet system were not successful. Systematic bias of this sort, if consistent, does not directly affect the determination of air pollution effects on children's health, because these determinations are made on the basis of relative differences in exposure.

The ambient field testing indicated that the TWS nitric acid measurements were 26 percent greater than nitric acid measured by the modified SCAQS sampler (by comparison 14-day integrated TWS data with the corresponding 14 sequential 24-hr samples). The reason for the positive bias in nitric acid concentrations was not determined. Gradual oxidation of collected nitrite compounds over 14-day time period was suspected as possible reason for the bias (Lurmann et al., 1994). Again, a systematic bias is not important for epidemiologic purposes if it is spatially and temporally consistent.

The accuracies of the TWS measurements for  $PM_{2.5}$  mass,  $PM_{2.5}$  chloride, hydrochloric acid, formic acid, and acetic acid were not determined in the field evaluation, but are expected to be comparable to other samplers using similar collection techniques. The one species of particular concern is acetic acid because the measurement technique is potentially subject to interferences and positive bias (Lurmann

et al., 1994) The acetic acid concentrations should be viewed as upper limits to the actual concentrations.

The precision of the TWS is an important characteristic for epidemiologic purposes. The initial objectives for precision were  $\pm 7$  percent for concentrations above the detection limit; this objective was more stringent than the general target of  $\pm 15$  percent for all exposure measurements. During ambient field testing, the TWS met the initial precision goals for nitric acid, fine particle sulfate and ammonium, while the precision for fine particle nitrate was about double the initial target value. Concentrations of hydrochloric acid and particulate chloride were too low to meaningfully characterize precision.

During the first 22 weeks of field sampling, data from collocated samplers show that precision targets were met for nitric acid and  $PM_{2.5}$  nitrate (see Table 3-10). The precision for  $PM_{2.5}$  mass,  $PM_{2.5}$  sulfate,  $PM_{2.5}$  chloride, hydrochloric acid, and formic acid were between  $\pm 7$  and  $\pm 14$  percent. Precision data for  $PM_{2.5}$  ammonium and acetic acid were not available because there were too few collocated samples with concentrations above the lower detection limits. Thus, the TWS precision for all of the chemical constituents for which precision was determined in Phase II was in agreement with the general target of  $\pm 15$  percent for all exposure measurements.

#### **QA Plan and SOPs**

Documentation of the TWS field operations and QA/QC procedures are described in the QA Plan (Peters et al., 1994) and the TWS QA document (Main et al., 1994a). The QA Plan includes detailed information on field operations in the sections titled: "Field Operations Program Plan for the Two-Week Acid/Aerosol Sampler" and "Field Operations Manual for the Two-Week Sampler". These documents serve as detailed operating SOPs and also address all of the QA elements identified in the introduction to this section except data management and data validation. The TWS data validation and data management are described by Lurmann et al., (1994). The data validation is straightforward because there are very few things that can go wrong in the TWS. The TWS data were considered valid unless there were sampler flow rate problems in the field, laboratory notes indicating invalid data due to analytical or other problems, or concentrations that were far outside of the expected ranges.

#### **QC, Validation, Data Results**

Data from blank filters and collocated samplers were regularly examined. Blank data were used to calculate final concentrations, and used to monitor field and laboratory performance. The collocated sampler results were used to monitor combined field and laboratory sampler precision during actual field use. The results were consistent with or better than performance expected on the basis of sampler development and testing data. Data validation checks during data processing includes screening and

flagging for various sampler or laboratory operational problems, as well as quantitative comparisons of sampler data with target ranges, among filters within a sampler, and among samplers.

Ambient TWS data were encoded in electronic spreadsheets by STI. The data were archived monthly and sent to the USC data manager after the first six months and then quarterly thereafter.

### **External Audits**

The TWS audit frequency was approximately every 6 months during Phase II. The audits consisted of observing sampler operations, checking the functionality of pressure and temperature sensors, and checking the sampler flow rates. Sampler flow rates were measured using an orifice calibrated against a wet test meter. The pressure drop introduced by the orifice is less than one inch of water to minimize disturbance to sampler flow rates. The orifice design was sensitive to fluctuations in ambient wind speeds. The audit flow rates were converted to flow rates at standard temperature and pressure using barometric and temperature readings observed during the audit. TWS Dwyer rotameter readings were recorded during the audit and converted to flow rates using calibration equations provided by the station operator. These readings were only used to provide immediate feedback on comparison of station and audit flow rates. The Dwyer rotameter readings were not used by STI to calculate TWS flow rates. The station flow rates used for formal comparison with audit values were the two-week average corrected flow rates for the relevant sample periods at each station. The two-week average corrected flow rates were based on initial, mid-sample, and final Gilmont rotameter readings. The average flow rate values provided by the STI data manager were already corrected to standard temperature and pressure (using an average of the initial, midpoint, and final temperatures, and the site specific average barometric pressure).

### **System Audit Results.**

CCC conducted a site visit in September 1993 when the TWS were undergoing initial field testing. The purpose of the visit was to examine the TWS configuration and assess the initial testing and operating procedures. The general conclusions of the site visit were that samplers were well-constructed and operating correctly, and that the design and implementation of the test experiments were sound, including sampler set up and use of LAREI laboratory to handle filter operations. Minor problems were overcome by implementing mechanical changes and/or monitored by incorporation of additional QC checks into routine operations. The results of the site visit and sampler evaluation indicated the samplers were ready for field deployment.

### **Performance Audit Results**

The results of a performance audit for the TWS sampler are shown in Table 5-3. The table shows the difference between the 2-week average flow rate in standard liters/minute (slpm) reported by STI, and the one-time audit measurement in slpm, reported as a percentage of the audit flow rate. The nominal flow rate values for channels A, B, and C are 0.4, 0.4, and 1.2 slpm respectively. STI determined reported slpm values from initial, midpoint, and final Gilmont rotameter readings, initial, midpoint, and final temperature readings, and average station pressure readings. All of the results are within the expected target range of  $\pm 10$  percent. The results indicate good agreement, especially considering that one instantaneous measurement was compared with a two-week average flow rate.

#### **5.1.3 Sampling at Schools**

The Timed Exposure Diffusion (TED) ozone samplers were utilized to measure ambient and in-classroom integrated ozone concentrations at selected schools. The TED samplers control the sampling time periods and air velocity across a passive sampling device (the Koutrakis passive ozone badge sampler). STI was responsible for field operation of the samplers, and LAREI was responsible for sampler badge assembly and disassembly, chemical analysis of the sampler badge filters, and reporting laboratory data to STI. STI was responsible for combining laboratory data with field data, calculating final concentrations, and reporting final data to the USC data manager.

### **Sampler Development and Testing**

The detection limits of the Koutrakis badge were fairly well known, and interference effects were partially understood at the beginning of the development effort. It was also known that air velocity influenced the collection rate of the badge. A TED system was designed and tested following a well-designed protocol. The sampler underwent extensive laboratory and field testing during the development and acceptance process to characterize interferences, detection limits, and precision of the overall system: TED sampler plus ozone badge plus LAREI laboratory analysis. The detailed explanations of the need for the new sampler, the rationale guiding its design, laboratory test results, field test results, and final precision and accuracy estimates are provided by Lurmann et al., (1994). Additional precision information was collected through deployment of collocated with continuous ozone monitors, and deployment of collocated TED samplers, trip blanks, and field blanks at selected locations. Some key features of TED sampler performance are briefly discussed below.

The accuracy of the sampler is affected by positive interferences from nitric acid and hydrogen peroxide. Since the concentrations of these species are small in comparison to ozone concentrations, the magnitude of the bias introduced by the

interferences is small. Field comparisons with SCAQMD operated continuous ozone analyzers at Azusa indicated +6 percent bias for the TED sampler.

The precision of the TED sampler is an important characteristic for epidemiologic purposes. The objectives for TED sampler precision was  $\pm 15$  percent. The precision of the sampler is affected primarily by blank variability. Since the sampler integrates exposure over time, long sampling times and/or high concentrations result in exposures well above blank levels. The minimum practical exposure time is set in conjunction with expected concentration levels to achieve precision and detection limits in the desired range. During the Azusa field testing a precision of  $\pm 12$  percent was achieved for 22-hour exposures. Field sampling at schools was conducted using 28-hour total exposure periods. Sampler precision and accuracy of the sampler were determined to be acceptable for use in the study.

### **QA Plan and SOPs**

Documentation of operations and QA/QC for the TED sampler ozone measurements is contained in the QA Plan (Peters et al., 1994), the TED sampler QA Plan (Main et al., 1994b), and Lurmann et al., (1994). The "Field Operations Program Plan for the Micro-Environmental Ozone Measurements in Schools" is incorporated in the QA Plan. This document serves as detailed operating SOPs and also addresses all of the QA elements identified in the introduction to this section, including data management and data validation. The TED sampler data management and data validation are described by Lurmann et al., (1994). They present a fairly detailed description of data processing and data validation.

### **QC, Validation, Data Results**

Data from trip blanks, field blanks, and collocated samplers were regularly examined by STI. Blank data were used to calculate final concentrations, and monitor field and laboratory performance. Collocated sampler results were used to monitor combined field and laboratory sampler precision during actual field use. The field results were consistent with performance expected on the basis of sampler development and testing data. The data validation checks included screening and flagging for various sampler or laboratory operational problems, as well as quantitative comparisons of sampler data with target ranges, comparisons among indoor and outdoor samplers, among samplers at different schools within a community, and comparisons with continuous ozone measurements in the community. The school ozone database was delivered to the USC data manager at the end of 1994.

### **External Audits**

Performance audits of TED field sampling were not performed. The only practical audit methods would be to use collocated samplers and/or collocated continuous



analyzers. Both of these activities were incorporated into STI's routine procedures. The performance audits of the laboratory operations are discussed in Section 5.1.4.

A system audit of TED sampler field operations was conducted by CCC during October of 1993. The general conclusions of this audit were that (1) the samplers were being deployed according to a well-designed protocol, (2) the field operations were being handled in an exceptionally efficient, professional manner designed to minimize any potential problems such as sample mix-ups or battery failure; (3) the operations incorporated sufficient QC data to characterize sampler precision and accuracy; and (4) the operations were weakly documented. Documentation of operations was subsequently developed. An important feature of the operations was the use of trip blank and field blank data to calculate final ambient concentrations.

#### **5.1.4 Laboratory Operations**

LAREI was responsible for laboratory operations in support of the TWS and TED sampling. Their responsibilities included TWS filter and denuder preparation, TWS and TED sample holder assembly and disassembly, sample extraction, chemical analysis of the sample extracts, and reporting of laboratory data to STI. The analyses included gravimetric determination of PM<sub>2.5</sub> mass for TWS samples, ion chromatographic (IC) analysis of nitrate, sulfate, chloride, ammonium, formate, and acetate ions for TWS samples, and ion chromatographic analysis of nitrate for TED sampling.

New equipment and procedures were needed at LAREI to conduct some of the laboratory analyses, particularly for formic and acetic acid. New sample handling and analytical procedures were developed concurrent with TWS and TED sampler development efforts. The new procedures were developed for ion chromatographic analyses for formate and acetate. Improved procedures were developed for ion chromatographic analyses of sulfate, nitrate, ammonium, and chloride.

#### **QA Plan and SOPs for Laboratory Operations**

LAREI developed a detailed SOP describing laboratory operations in support of each aspect of TED and TWS sample handling and analysis. They also developed a more general SOP describing standards, controls, blanks, and QC criteria for analytical runs. A QA Plan addressing more general issues such as project management, reporting schedules and content, general descriptions of methods and limitations etc. was under development at the end of Phase II.

#### **QC, Validation, and Data Reporting**

QC data generated by LAREI includes lab blanks, calibration standards, laboratory control standards, and replicates. LAREI was responsible for interpretation and reporting of this QC data. LAREI did not interpret results for trip blanks, field blanks,

or collocated samplers. The analytical results from these field QC samples were reported to STI for interpretation. Data from analytical runs were reviewed by the laboratory manager for satisfactory calibration and QC sample performance before reporting to STI. Certain QC data were plotted for easier monitoring of performance. The QC data were reported to STI along with the sampler and analytical data.

Sample preparation and analysis schedules for the TWS were designed to follow the two-week sampling schedule, since it is desirable to avoid prolonged storage of the active sample media used in the TWS. Likewise TED sampler analysis schedules called for quick turnaround to avoid oxidation of nitrite to nitrate on the filter. Laboratory data were reported in hard copy form to STI shortly after generation at the laboratory. Typically, STI received the analytical data three to four weeks after the samples were submitted to LAREI.

Backlogs in analysis and reporting occurred on occasion at LAREI. However, the Phase II TWS data were usually analyzed within four weeks of receipt by the laboratory.

### **External Audits**

External audits of laboratory operations were scheduled to occur once per year. A system audit was completed during October 1993, and a performance audit was conducted at the end of 1994.

The system audit of laboratory operations was conducted in October 1993, when many procedures were still under development. The general results of that audit were that laboratory staff and management were capable, ongoing laboratory operations were handled in accordance with good practice, new sample handling and analyses procedures were being developed in accordance with good practice and in accordance with significant technical input from the TWS and TED sampler developers, and adequate QC was being incorporated into the procedures. Documentation of operations were still being developed; responsibilities and procedures for data quality assessments and reporting were not defined. The documentation of operational procedures was subsequently prepared. However, the procedures and documentation for data quality assessments were still in need of improvement at the end of Phase II.

A performance audit of the laboratory operations was conducted by CCC. Prepared filters supplied by LAREI were spiked by CCC and returned to LAREI for analysis in 1994. The spikes included PM<sub>2.5</sub> mass, nitrate, sulfate, chloride, ammonium, formate and acetate. The results indicated good performance for PM<sub>2.5</sub> mass on Teflon filters and for nitrate, sulfate, chloride, ammonium, formate and acetate on carbonate filters.

## **5.2 PULMONARY FUNCTION TESTING QUALITY ASSURANCE**

USC is responsible for administration of pulmonary function tests. LAREI is responsible for instrument calibration and QA/QC. A QA Plan addressing each of the QA elements identified in the introduction to this section has been developed. SOPs and a Health Assessment Operations Manual that provide detailed descriptions of field and laboratory operations have been prepared and are in use. These procedures include abundant generation of QC check data. External audit activities including field site audit visits, calibration checks, and evaluation of instrument and technician performance through the QC data are conducted and reported regularly. The following is a discussion of QA/QC activities and results.

### **5.2.1 Introduction**

As stated in the original proposal, the broad goals of this effort are to:

(i) Assure cross-sectional comparability (Spirometers' and test technicians' performance must not change from one school or community to another; any observed difference between sites must reflect true lung function differences).

(ii) Assure longitudinal comparability (Spirometer or technician performance must not change from one year to the next; any observed differences over time must reflect true lung function growth).

(iii) Assure external comparability (Lung function measurements should be directly comparable with those in other projects, particularly the Harvard 6- and 24-city studies, to test the possibility of generally lower function in Southern California than elsewhere).

In Phase II, goals (i) and (ii) have been achieved to a high degree, as explained in the remainder of this section. A cross-comparison of CARB/USC equipment with Harvard equipment to meet goal (iii) has been postponed because the anticipated local visit of the Harvard testing team (to Simi Valley) did not take place. This delay is of little concern, because comparisons between our data and Harvard's will not become important until a fairly late stage in the analysis of both. Once a cross-check is performed, valid data adjustments should be possible both retrospectively and prospectively.

Compared to clinical practice or smaller-scale air pollution/health research, this project demands a much higher degree of accuracy and precision in lung function testing, for two reasons. First, a large subject population provides high statistical power to detect small function differences between communities -- either real (due to exposures) or spurious (due to inadequate standardization of testing). Second, even the smallest detectable real differences in function or growth rate may have important long-

term health consequences. Such differences are usually not important in smaller-scale research or clinical testing, and even the best commercial testing equipment is not necessarily designed to detect them. The American Thoracic Society's standards [ATS, 1987] allow individual spirometers to differ from a standard reference instrument by plus or minus 3 percent in their volume measurements -- adequate accuracy for most clinical applications. Thus in principle, the highest and lowest ATS-acceptable measurements of a given individual's vital capacity may differ about 6 percent. But in typical middle-aged adults the loss in vital capacity with aging is roughly 1 percent per year. Decreased vital capacity is statistically associated with shortened life expectancy. No one knows for certain what causes this association, or who among the low-vital-capacity population is at risk. In the absence of more specific knowledge, we must assume that even a decrement in average vital capacity as small as 1 percent in a polluted community (relative to an otherwise similar clean community) predicts excess chronic illnesses and early deaths. To be able to detect average function decrements that small, we must insure that measurement differences across space and time are much smaller than permitted by ATS standards.

Consistently high precision and accuracy cannot be guaranteed for any single piece of equipment or person over the long times and distances involved in this project. The practical goal must be to minimize the contributions of each individual spirometer, each individual technician, and each unique set of conditions at a particular time and test site, to the overall variance of lung function data. This goal is approached (i) by calibrating all pieces of equipment and coaching all technicians to a highly uniform performance level before they are put to work gathering data; (ii) by having technicians check calibration of their equipment frequently during actual testing, and having the spirometry QA officer review their work regularly; and (iii) by using results from (ii) to guide post-hoc statistical adjustments of lung function data which compensate for unavoidable small variations in performance between different pieces of equipment, and over time in any one piece of equipment.

Spirometers have undergone multiple daily calibration checks in the field, and semiannual laboratory calibrations against volume and time standards. Technician-related and spirometer-related variability has been monitored regularly by the spirometry QA officer, who copies computer data files from each system during each field QA inspection and reviews them later. The initial five technicians and field team leader underwent a training course covering spirometer calibration checks, coaching and motivation of subjects, and recognition of unsatisfactory test maneuvers, conducted by senior investigators and the QA officer. The initial staff also conducted a preliminary practice field testing session at a school not involved in the main study. Three replacement technicians, one of whom later became the replacement field team leader, have been trained on the job by their fellows. As Section 5.2.4 indicates, their measured performance has been comparable to that of the original team members.

### 5.2.2 Spirometry Equipment Quality Assurance Procedures

Hardware and software used for testing students' lung function and for data storage and retrieval were essentially the same as in the Harvard 24-City Study. Before field testing began, our investigators met with field testing and data management supervisors from the Harvard School of Public Health to verify that we understood their procedures and could substantially duplicate them. For an independent test of each system's performance, all six field spirometers (P.K. Morgan Ltd., Gillingham, UK) and their associated personal computer hardware and software were taken to the spirometer testing laboratory at LDS Hospital, Salt Lake City (Robert Crapo, MD, Director). The lab staff, in cooperation with our representative, tested each system against the 24 ATS standard waveforms [ATS, 1987]. A slight discrepancy in volume readout between our systems and the LDS waveform generator was noted, and an independent reference standard was not available. Thus it was not possible to obtain an "absolute" independent volume calibration during this testing. Nevertheless, the more important component of the testing was successful: the mechanical performance of each spirometer and the electronic performance of its digitizing and computing system was verified to be satisfactory. The necessary volume calibrations are performed periodically at our Rancho Los Amigos Medical Center (RLAMC) reference laboratory. Twice a year (shortly before and shortly after the field testing season) and immediately after any repair service, each spirometer system is tested to verify its volume and flow rate readings. This testing employs independently verifiable physical measurements or standard electronic testing instruments, not dependent on any spirometric device [Linn et al., 1990]. Volume and flow readings from each field instrument are compared against a laboratory reference spirometer -- a Stead-Wells type low-inertia water-sealed spirometer with accessories for precise digital voltage readout of volume (i.e. spirometer bell position) interfaced to a personal computer. Its characteristics of volume change vs. voltage output change are determined by withdrawing volumes of air using a water displacement apparatus. The displaced water is weighed accurately with a laboratory balance, each 1 g of water representing 1 ml of air removed from the spirometer. Flow measurements from the reference spirometer are computed in terms of volume change per unit time, time measurements being provided by the computer's crystal-controlled clock. Clock frequency is in turn verified with a conventional electronic frequency counter traceable to a manufacturer's standard. The reference spirometer is used in conventional mode to test the readings of calibration syringes (described below), which are in turn used to test the readings of field spirometer systems both in laboratory tests and in routine field calibration checks. In additional lab tests, the reference spirometer is connected directly to each field spirometer and operated in "reverse" mode (with volume signal polarity reversed so that air leaving the reference spirometer and entering the field spirometer yields a conventional positive volume reading on both). For each such test the reference spirometer bell is raised to a standard height, then released and allowed to fall under the influence of gravity. Standard weights of different magnitude are placed on the bell to provide different flow rates.

Three-liter calibration syringes with electronic volume and flow readouts (Jones Medical Instrument Co., Oak Brook, IL) are kept with spirometers continuously in the field, to avoid any errors due to temperature discrepancies between syringe and spirometer. A syringe is used to check volume calibration at the beginning and end of each field testing session, and just before and after any intermission. The syringe's volume and flow readings, along with the time of testing, are entered into the electronic database along with the spirometer's corresponding readings. Before and after each field testing season, each syringe's average volume output is measured to the nearest 0.01 liter, by repeated injections into the laboratory reference spirometer which has recently been calibrated against the aforementioned independent standards. Different syringes may vary by at least 0.03 liter in delivered volume. Ideally, these variations will be reflected by equivalent differences in the syringes' electronic readouts; if not, the remaining difference are taken into account in calibration procedures to minimize differences among spirometers. As explained later, field and laboratory data agree that syringes' electronic readouts faithfully reflect small changes in volumes actually delivered, and thus provide the most reliable basis for adjustment of subjects' spirometric data.

Temporal and spatial variations in ambient temperature and barometric pressure importantly influence spirometric results; therefore they must be accounted for very precisely. This is conventionally done through the BTPS (body temperature and pressure saturated) correction, by which measured volumes and flows are always expressed in terms of air saturated with water at 37° C and one atmosphere. The spirometry software includes a BTPS correction algorithm; the only input required from the technician is the correct ambient temperature and absolute barometric pressure. A precision digital thermometer is mounted on each spirometer case. The technician reads the thermometer frequently during a testing session and updates the temperature entry in the software whenever the thermometer reading changes by 0.2° C or more. This limits temperature-related variation in spirometric measurements to the order of 0.1 percent.

Short-term variations in barometric pressure influence spirometric results far less than temperature variations, and are therefore ignored. Effects of pressure change with altitude are important, however, since the test communities' altitudes range from 10 to about 1,500 m above sea level. Accordingly, prior to field testing a senior investigator made multiple pressure measurements in each community with a precision aneroid barometer and compiled a table of typical barometric pressures for each community. Technicians refer to the table and update the barometric pressure entry in the spirometry software immediately upon moving from one community to another.

The temperature and pressure employed in the BTPS correction for every spirometric test are stored in the electronic database to allow checks for technician errors. A wrong barometric pressure entry is detected and corrected simply by reference to the community code. Overall validity of temperature data is checked by regression analysis testing the relationship of measured vital capacity to temperature after

accounting for the usual predictors -- age, size, sex, and race. Results should show no dependence on temperature. Isolated substantial errors in temperature input are detected as outliers -- individual temperature inputs differing noticeably from preceding and following values on the same system, or differing from nearly simultaneous inputs on different systems in the same location.

### **5.2.3 Technician/Subject Performance Quality Assurance Procedures**

Instructions given by investigators to test technicians, and by technicians to subjects, are based on published recommendations [Enright and Hyatt, 1987] and on the Harvard 24-City Study field testing manual. To help maintain technician performance as well as to check on problems with equipment, the spirometry QA officer made unannounced visits to each test community at least once in each season throughout Phase II, observing all aspects of function testing and interviewing each technician to learn of any operational or equipment problems. Technicians inevitably have some awareness of pollution levels in each community. This potentially could bias results; e.g., if a technician is more inclined to accept slightly submaximal test performances in more polluted communities. Technicians have been instructed specifically to guard against this possibility. The proportion of accepted versus rejected test maneuvers has been analyzed to assure that it does not vary substantially between communities.

The P.K. Morgan/Harvard software automatically measures each blow with respect to three ATS criteria -- FEV<sub>1</sub>, extrapolation volume (low values representing appropriately fast acceleration of the expiratory muscles at the beginning of the blow), last-2-seconds volume (low values indicating complete expiration), and forced expiratory time (high values indicating complete expiration). The software also assigns a "computer code", rating the blow acceptable if the aforementioned three variables are within ATS-specified limits. The technician assigns a separate "technician code" of acceptability judged from observation of the subject and the tracing, as well as a comparison with any previous blows by the same subject, automatically provided by the software. ATS criteria, mainly addressed to testing of adults with respiratory disease, reject some blows from youngsters which are in fact good representations of their pulmonary physiology. Accordingly, the percentage of "technician acceptable" blows exceeds the percentage of "computer acceptable" blows. As discussed elsewhere, the primary criteria of data acceptance for statistical analysis are technician acceptance, and reproducibility of two or more blows according to criteria adapted from the Harvard studies.

"Technician performance statistics" -- percentages of accepted vs. unaccepted blows, means and distributions of the three ATS criteria variables -- have been compared among individual technicians repeatedly during each testing season, on the basis of data collected by the QA officer during field visits. Feedback was provided to the individual technician and team leader when these statistics suggested a problem. (Only minor problems were found, and only rarely, during Phase II). These human

performance statistics of course reflect the behavior of subjects as well as technicians. Even the best technician cannot obtain acceptable blows from subjects unable or unwilling to perform them. Technicians' differences in performance statistics depend partly on their different rates of encountering "unsatisfactory" subjects. To avoid bias, "unsatisfactory" subjects should arise only rarely and should be evenly distributed among communities and among technicians. This cannot be assured, however, because logistic practicalities do not allow a perfect geographic and demographic balance of subjects across technicians. (At least one potential bias must be introduced deliberately: Spanish-only-speaking subjects must be assigned to Spanish-speaking technicians). Appropriate statistics have been calculated to verify that these biases are small and unlikely to affect overall conclusions (see Section 5.2.4.3).

## **5.2.4 Results from Phase II Field and Laboratory Testing**

### **5.2.4.1 Comparison of Lab Reference Spirometer to Water Volume Standard**

Successive water displacement tests yielded the following calibration factors (in ml per unit increment of digitized voltage signal): 2.079 in January 1991 and again in February 1992, 2.095 in July 1993, 2.127 in December 1993, and 2.102 in July 1994. The increment of less than 0.8 percent observed in July 1993 was considered non-significant. Thus the calibration factor in the reference spirometer's software was not changed at that time, i.e. it was the same for laboratory calibrations with the reference spirometer both before and after the 1993 field testing season. The further increment of about 1.5 percent observed in December 1993 was considered significant, so the newly determined calibration factor was incorporated in the reference spirometer's software. Following this change, the reference spirometer read out volumes of Jones syringe injections equal to or a fraction of a percent larger than the syringes' own volume readouts, whereas previously the spirometer had read about 1 percent less than the syringes. Given that this shift occurred with all four syringes, it seems likely that syringes' volume readouts did not change appreciably, relative to their actual volumes delivered. Rather, the reference spirometer's reading of syringe outputs changed because of the change in the reference spirometer's calibration factor. In other words, volume readouts from the reference spirometer (as calibrated periodically by changes of water volume) appeared to be less reproducible than volume readouts from the Jones syringes. Nevertheless, the reference spirometer and water displacement procedure still appear useful to guard against calibrations drifting over long time periods. In light of the above findings, the calibration factor in the reference spirometer's software was not changed after the final water-displacement test in July 1994, although the result was 1.2 percent below that from 7 months earlier (still 0.3 percent above that from one year earlier).



#### 5.2.4.2 Comparisons of Reference Spirometer, Syringes, and Field Spirometers

The upper graph of Figure 5.2.1 shows results of calibration checks at the end of the 1993 field testing season involving multiple 3-liter injections from each syringe into each field spirometer. Each vertical bar indicates the mean ratio of the syringe's electronic readout of FVC to the spirometer/computer system's corresponding readout, for a given syringe/spirometer pair. Differences among spirometers were highly significant ( $P < 0.00005$  by analysis of variance) and far exceeded differences among syringes (not significant by analysis of variance,  $P > 0.1$ ). Analogous tests of FEV<sub>1</sub> and maximal midexpiratory flow rate data gave similar results. At the end of the 1994 testing season, findings were generally similar. The pattern of "high" and "low" FVC readings among spirometers was different from that of 1993 -- an expected result of intercurrent laboratory calibrations and possible spontaneous shifts (see below). In checks at the end of the 1994 season, variation among syringes reached statistical significance, but was still far smaller than variation among spirometers.

The center graph of Figure 5.2.1 shows results of syringe injections into the laboratory reference spirometer before and after the 1993 field testing season. As discussed in Section 5.2.4.1, the reference spirometer read out consistently lower volumes than did the syringes themselves. Syringe and reference spirometer readouts agreed that Syringe 1 increased its delivered volume significantly (by more than 1 percent), Syringe 2 decreased its delivered volume by a similar or slightly smaller amount, and the other two syringes changed little. Minor mechanical problems were found in Syringes 1 and 2 at the end of the testing season and were repaired. The problem with Syringe 2 appeared to explain its changed output; for Syringe 1 the cause of changed output was uncertain. The lower graph of Figure 5.2.1 shows syringes' daily mean FVC readouts from calibration checks throughout the 1993 field testing season. These data tended to corroborate the laboratory tests of syringes, showing a persistent upward shift in midseason for Syringe 1, a persistent downward shift late in the season for Syringe 2, and no appreciable change for the other syringes. Thus both laboratory and field data from 1993 supported the conclusion that the syringes' electronic readouts are the most reliable component of the field system for volume measurement. Comparisons of pre- and post-season calibration checks in 1994 showed a mean decrease of 52 ml in the readout from Syringe 1, with a corresponding mean decrease of 42 ml in the reference spirometer's readout. Syringe 2 showed a mean increase of 16 ml in its readout, with a corresponding mean increase of 13 ml in the reference spirometer's readout. Syringes 5 and 6 showed no change pre- to post-season in their own readouts, and mean increases of 11 ml and 5 ml respectively as read by the reference spirometer. Thus the 1994 results were similar to 1993 results except that when syringes appeared to change their output volumes, they did so in the opposite direction. Syringes 3 and 4, acquired during the 1994 testing season, fell within the range of the other syringes in terms of their relationships of volume readout to volume delivered.

Field spirometers' and syringes' flow measurements could not be independently checked in the same manner as volume measurements. However, the observed tendency for FEV<sub>1</sub> and MMFR variation to track FVC variation argues that flow-related as well as volume variables are affected by whatever factors cause inter-spirometer differences.

One probable cause of inter-spirometer differences is unavoidable slight variations in the Morgan spirometer laboratory calibration procedure. In addition, the spirometers exhibited small but meaningful shifts in output during the testing season, which coincided with moves from one community to another and may reflect physical shocks during the moving process. Figure 5.2.2 illustrates this phenomenon. The upper graph shows an upward shift in a spirometer's calibration-check FVC reading, similar in injections with two different syringes, and not explainable by the changes in syringe volume output shown in Figure 5.2.1. This shift was reversed promptly by running the Morgan laboratory calibration procedure at the field site. The lower graph shows a similar shift by a different spirometer at a different time. In this case there was no intervention, and the shift persisted. In light of these observations of meaningful change in spirometers' volume readouts, contrasting with the relatively greater stability of syringe volume readouts, we elected to adjust every subject's spirometric data on the basis of the last preceding and first following syringe calibration check data. A small percentage of calibration checks were invalid due to technician errors or temporary malfunctions of equipment. These were recognizable by large inconsistencies of syringe and spirometer volume readings, and were not used in data adjustments.

All of the aforementioned variability of field testing equipment was within the range expected a priori (see Section 5.2.1). That is, variations were too small to be of concern to typical users of such equipment, and did not reflect important design or manufacturing problems, but still might have introduced important bias into our statistical tests for air pollution effects had they been ignored. The major unanticipated finding was the high precision of syringe volume readouts, which allowed us to use them as a "gold standard" for detecting small variations in spirometer readouts, and thereby to measure subjects' lung function with more consistency than we could have with spirometers alone, or with spirometers plus non-electronic calibration syringes.

#### 5.2.4.3 Technician/Subject Performance

The spirometry QA officer visited all 12 communities each season as planned, observing a few testing sessions and copying all spirometry data then resident on each computer. Extra visits were made only to the nearest community (Long Beach) because of the costs involved. Occasional minor problems of equipment malfunction or misidentification of stored data were noted at these visits. Problems were reported to senior investigators for future correction, or corrected on the spot when feasible. Some individual technicians were counseled on occasion to correct minor flaws in their test procedures as revealed by technician performance statistics. When new field technicians (Numbers 9-11) first began to collect data, their performance statistics were given special attention in the interim data analyses in order to detect any deviation from the experienced staff. No such problems were found.

At the end of each year's testing, comprehensive performance statistics were calculated for each field technician. Table 5-4 shows results. The only individual with a markedly low percentage of acceptable blows was Number 7, a senior investigator who substituted occasionally when one of the regular technicians was absent, and performed fewer than 2 percent of all tests each year. It is not clear whether these statistics represent effects of limited irregular testing experience, or chance encounters with a high percentage of difficult subjects, or both. Differences between 1993 and 1994 reflect the change in protocol. In 1993, the practice was to stop testing after the seventh blow or the third technician- and computer-accepted blow, whichever came first. In 1994, seven blows were obtained routinely to give each subject the same a priori opportunity to maximize performance. Accordingly, the percentage of technician-accepted and computer-accepted blows rose in 1994. Overall averages for specific performance variables from technician-accepted blows also improved slightly in 1994. Each technician's average for these variables met the American Thoracic Society's acceptable limits, but all technicians accepted some blows outside the ATS acceptable range. This was appropriate because, as discussed in Section 5.2.3, ATS criteria are too stringent for tests of young, generally healthy subjects.

Performance statistics also were calculated for each separate community, to address the possibility that intercommunity performance differences might be mistaken for pollution effects. As Table 5-5 shows, intercommunity differences were consistently small. Differences in average performance statistics among subjects of different genders, ages, or races were also examined using analysis of variance, and found to be generally small. Most of the tested differences were statistically significant, however, due to the large number of data points and consequent high power of statistical tests.

To verify that technicians consistently readjusted their spirometers to compensate for ambient temperature, multiple regression analyses were performed part way through the 1993 testing season. Forced vital capacity was the dependent variable. Temperature was included as an independent variable, along with the usual predictors size, gender,

and age. Each analysis was limited to one race and grade level to avoid a need for more complicated regression models. Temperature was consistently non-significant as a predictor of forced vital capacity, indicating that the data were not biased appreciably by any errors of temperature compensation.

#### **5.2.5 Recommendations Concerning Spirometry Quality Assurance**

Given the apparent success of the spirometry quality assurance effort, it is recommended that procedures be kept basically the same. However, it seems appropriate to redistribute the effort toward more interim data analysis and fewer field visits. The most important guarantor of quality is the recording and analysis of technician performance data and calibration data. Field inspections are considered useful to maintain staff vigilance. However, much less than 1 percent of field testing is actually observed, so the chance of uncovering an otherwise unrecognized problem during an inspection visit is small. Also, most of the necessary time and expense relates to travel rather than actual field observations. Accordingly, it seems reasonable to require only two inspections within three years in a given community, rather than one per year. The requirement for more than one inspection per year in at least one community should be retained, so that technicians may expect a visit at any time. With this schedule, analysis of data collected at one visit can be completed routinely before the next visit, and any necessary feedback to the field staff or investigators can be provided more consistently.

### **5.3 QUESTIONNAIRES AND ABSENCE MONITORING**

USC was responsible for development, administration, and data processing of questionnaires, telephone inquiries, and absence monitoring. A QA Plan addressing each of the QA elements identified in the introduction to this section was developed. SOPs describing questionnaire data processing were developed. Data processing operations for questionnaires include formalized procedures for verification of data entry and for detection and flagging of unreasonable responses. Data processing and submission to the overall data manager have proceeded apace with operations.

The results of questionnaire administration are communicated quarterly to the investigators. Analyses of the return rates and the responses to individual questions are used to guide development of modifications to the questionnaires. The questionnaire manager utilizes these analyses, plus consultation with outside experts, consultation with the relevant investigators, and pilot tests to develop modifications intended to increase the utility of the information obtained.

### **5.4 DATA MANAGEMENT**

Data management and data validation responsibilities were distributed among the operational components of the study, as well as flowing to the final or overall data

management system at USC, where all study data will be archived and made available for distribution. Field and laboratory data for air quality monitoring flow to STI. STI was responsible for combining field and laboratory data, calculating and validating final concentrations, obtaining data from district operated stations, and for reporting final air quality data to the overall data manager. STI was also responsible for reviewing time/activity data from USC, generating air pollutant exposure estimates, and reporting these to the overall data manager. Field spirometry data, questionnaire data, and absence monitoring data flow directly to the overall data management system at USC. Thus the data manager at USC fills two roles: first, handling the routine collection, processing, and validation of health and activity related data; and second, the collection, archiving, distribution, and statistical analysis of final study data.

### **QA Plan and SOPs for Data Management**

The data management efforts at USC were mostly directed toward the collection and validation of health assessment data generated by USC personnel, and toward preliminary statistical analyses of these data in combination with historical air quality measurements supplied by STI. A QA Plan was developed that summarizes data management operations at USC and that addresses the elements of QA listed in the introduction to this section. The QA Plan provides an overview of operations but there is no documentation equivalent to an SOP that formalizes operations and provides sufficient detail to conduct the operations. This reflects the fact that a small group of in-house staff work closely together on data submissions, editing, validation, and data analyses. In order to make the data more accessible to outside parties, and to assure continuity of operations in the event of personnel changes, more detailed descriptions of the database contents, data validation, and editing criteria are being developed.

The QA Plan does not address the schedules and mechanisms for data submissions, data requests, or generation of statistical summaries and analyses. These activities are handled on an as needed basis by the data management staff, and have proceeded in a timely fashion. In order to assure timely availability of basic data sets and timely monitoring of study progress, schedules for data submissions, data capture/data validation reports, and analytical summaries should be established.

### **QC, Data Validation, and Reporting**

Though not formally documented, data editing, validation, and reporting to requesting parties have proceeded apace with data submissions. Data validation procedures include double entry and reconciliation of all manually keypunched data. Validated data subsets are screened for outlier criteria as needed to perform various analyses (no original data are discarded). The screening criteria are developed on an ad hoc basis to meet the needs of the analysis. Data quality assessment activities have included preparation of: spirometer and technician performance statistics evaluated by LAREI; questionnaire data reports used to develop revisions to the questionnaires;

subject retention statistics used to track continuity of subject participation with respect to study goals; and preliminary regressions to assess statistical power and to evaluate the usefulness of various information fields in the questionnaires.

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Table 5-1 Summary of Audit Results for Ozone and NO<sub>2</sub>.

Station	Operating Agency	Auditing Group	Date	Average Percent Difference	
				Ozone	NO <sub>2</sub>
Alpine	SDAPCD	SDAPCD	12/20/93	3.0	-3.3
Alpine	SDAPCD	ARB	8/12/94	3.0	-3.3
Atascadero	SLOAPCD	ARB	5/23/94	-4.9	-3.3
Atascadero	SLOAPCD	ARB	9/13/94	6.2	NA
Lancaster	SCAQMD	SCAQMD	11/30/93	NA	-7.1
Lancaster	SCAQMD	ARB	8/18/94	-10.7	-0.3
L. Arrowhead	STI	ARB	8/24/94	-5.6	-0.6
L. Elsinore	SCAQMD	SCAQMD	7/13/93	-1.8	4.6
L. Elsinore	SCAQMD	ARB	8/15/94	3.2	NA
Lompoc	STI	CCC	2/7/94	-1.5	-2.1
Lompoc	STI	ARB	8/9/94	-2.2	-4.8
Long Beach	SCAQMD	ARB	6/29/92	-2.7	-6.1
Long Beach	SCAQMD	SCAQMD	12/10/93	-3.7	1.7
Long Beach	SCAQMD	ARB	8/17/94	-11.3	3.5
Mira Loma	STI	CCC	2/8/94	-6.3	-2.9
Mira Loma	STI	ARB	8/23/94	-2.5	NA
San Dimas	STI	CCC	2/9/94	-1.5	1.7
San Dimas (transfer std)	STI	CCC	2/9/94	-2.7	-10.1
San Dimas	STI	ARB	8/25/94	-9.7	NA
Santa Maria	ARB	ARB	9/7/94	-0.8	1.3
Santa Maria	STI	ARB	9/9/94	-3.7	
UC Riverside	STI	CCC	2/4/94		1.7
UC Riverside	STI	ARB	8/29/94	-5.8	-4.2
Upland	SCAQMD	SCAQMD	2/11/94	-2.6	-2.2
Upland	SCAQMD	ARB	8/16/94	-9.5	-2.3
Upland	SCAQMD	ARB	8/26/94	-8.3	
					0.1

Table 5-2 Summary of Performance Audit Results for TEOM Flow Rates.

Station	DATE	auditing agency	total flow	main flow
Alpine	8/11/94	ARB	15.4	-0.3
Atascadero	8/10/94	ARB	1.3	-3.2
Lancaster	8/18/94	ARB	18.8	-3.8
L. Arrowhead	8/24/94	ARB	0.7	0.7
L. Elsinore	8/15/94	ARB	15.4	-4.7
Lompoc	2/7/94	CCC	5.6	4.2
Lompoc	8/10/94	ARB	1.4	-3.6
Long Beach	8/17/94	ARB	1.1	0.3
Mira Loma	2/8/94	CCC	13.9	-3.8
Mira Loma	8/23/94	ARB	5.2	-3.5
San Dimas	2/9/94	CCC	12.2	5.2
San Dimas	8/25/94	ARB	9.5	-5.4
UC Riverside	2/4/94	CCC	NA	5.5
UC Riverside	8/29/94	ARB	12.7	-4.5
Upland	8/16/94	ARB	12.8	-4.8

Table 5-3 Summary of Audit Results for TWS Flow Rates.

Station	sampler	date	channel A	Channel B	channel C
Alpine	5	12/8/94	1.6	1.1	-0.8
Alpine	17	12/8/94	-3.1	-1.5	-4.9
Atascadero	4	12/9/94	-1.4	0.9	1.5
Lancaster	13	2/9/94	-4.3	-2.4	-0.9
Lancaster	14	12/14/94	-2.8	-2.7	-0.3
L. Arrowhead	6	9/15/94	5.5	2.7	-1.4
L. Elsinore	8	12/8/94	-2.7	-2.6	-5.8
Lompoc	8	2/7/94	-2.2	-2.9	-0.4
Lompoc	3	12/9/94	-0.8	-1.2	-4.5
Long Beach	3	2/9/94	1.0	0.7	-2.2
Long Beach	1	12/14/94	-2.0	-2.0	-3.9
Long Beach	11	12/14/94	0.3	-1.3	-4.8
Mira Loma	7	2/8/94	-1.4	-8.4	-4.2
Mira Loma	16	12/15/94	-1.3	-2.0	-4.5
Mira Loma	?	12/15/94	-2.0	-2.0	-4.1
San Dimas	11	2/9/94	-1.9	-2.9	-5.1
San Dimas	14	2/9/94	-3.1	-1.0	-.28
San Dimas	12	12/15/94	2.0	0.0	-2.5
Santa Maria	2	12/9/94	-0.8	-1.0	-3.5
UC Riverside	12	2/4/94	-0.6	-1.3	-1.9
UC Riverside	16	2/4/94	0.3	-0.3	-2.0
UC Riverside	7	12/15/94	-1.3	-3.6	-3.5
Upland	9	12/15/94	0.3	-2.9	0.5

TABLE 5.4 TECHNICIAN/SUBJECT PERFORMANCE STATISTICS BY TECHNICIAN AND YEAR

TECH.	MEAN BLOWS/SUBJECT		% TECHNICIAN ACCEPTED		% COMPUTER ACCEPTED		% COMPUTER ACCEPTED*		EXTRAPOLATION VOLUME, MEAN $\pm$ SD*		LAST-2-SEC VOLUME, MEAN $\pm$ SD*		FORCED EXPIR. TIME, MEAN $\pm$ SD*	
	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994
1 (a)	5.98	-	69.3	-	41.0	-	59.0	-	4.4 $\pm$ 2.1	-	31 $\pm$ 172	-	6187 $\pm$ 1506	-
2	5.88	6.96	68.5	79.9	44.7	64.9	64.8	80.6	4.3 $\pm$ 1.9	3.7 $\pm$ 1.8	49 $\pm$ 389	3 $\pm$ 37	7218 $\pm$ 2175	7189 $\pm$ 1779
3	5.70	6.95	82.1	92.0	49.4	64.0	59.9	69.5	4.2 $\pm$ 2.5	3.9 $\pm$ 2.3	72 $\pm$ 300	37 $\pm$ 251	6620 $\pm$ 2342	7709 $\pm$ 2431
4	5.82	6.98	66.4	80.0	46.5	61.7	69.6	76.5	3.9 $\pm$ 1.9	3.8 $\pm$ 1.8	35 $\pm$ 222	9 $\pm$ 43	5813 $\pm$ 1282	6443 $\pm$ 1291
5	6.01	6.99	58.1	75.6	39.9	56.5	67.3	73.9	4.1 $\pm$ 2.0	3.8 $\pm$ 1.7	16 $\pm$ 72	10 $\pm$ 33	6785 $\pm$ 1305	5980 $\pm$ 1102
6	5.92	-	76.2	-	40.3	-	52.6	-	5.0 $\pm$ 6.1	-	66 $\pm$ 371	-	5935 $\pm$ 1387	-
7 (b)	6.50	6.89	46.4	35.3	29.4	37.1	58.3	87.3	4.6 $\pm$ 2.1	2.9 $\pm$ 1.2	13 $\pm$ 45	18 $\pm$ 108	7121 $\pm$ 1105	7127 $\pm$ 946
9 (c)	5.38	6.99	74.5	79.3	55.7	64.9	74.4	80.2	3.9 $\pm$ 1.7	3.7 $\pm$ 1.7	13 $\pm$ 127	6 $\pm$ 59	6780 $\pm$ 1600	6568 $\pm$ 1310
10	-	7.00	-	89.6	-	63.5	-	70.8	-	4.0 $\pm$ 2.1	-	15 $\pm$ 69	-	6237 $\pm$ 1109
11	-	6.99	-	75.3	-	62.9	-	73.9	-	3.6 $\pm$ 1.5	-	2 $\pm$ 28	-	6254 $\pm$ 1401
ALL	5.84	6.98	69.8	80.5	45.2	62.0	64.0	76.2	4.2 $\pm$ 2.7	3.8 $\pm$ 1.8	41 $\pm$ 260	11 $\pm$ 90	6512 $\pm$ 1817	6595 $\pm$ 1574

\*Considering technician-accepted blows only.

(a) Team leader 1993.

(b) Senior investigator substituting occasionally for absent regular technicians.

(c) Team leader 1994.

FEV<sub>1</sub> extrapolation volume in percent, last-2-seconds volume in ml, forced expiratory time in milliseconds. American Thoracic Society Guidelines: extrapolation volume < 5.0%, last-2-seconds volume < 200 ml, forced expiratory time > 6000 msec.

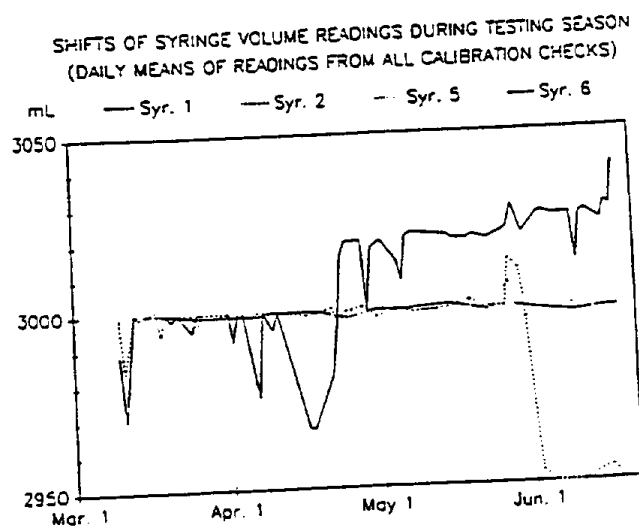
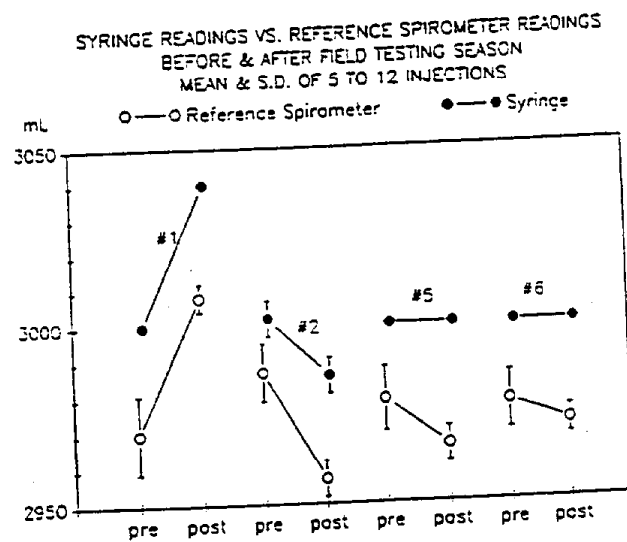
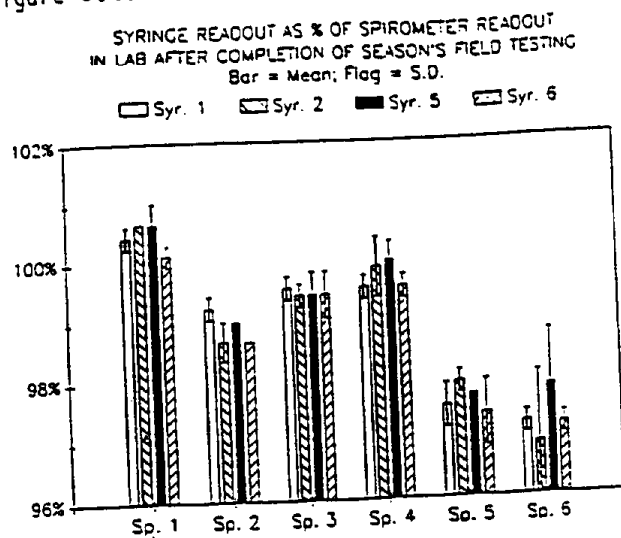
TABLE 5.5 TECHNICIAN/SUBJECT PERFORMANCE STATISTICS BY TOWN AND YEAR

TOWN	MEAN BLOWS/SUBJECT		% TECHNICIAN ACCEPTED		% COMPUTER ACCEPTED		% COMPUTER ACCEPTED*		EXTRAPOLATION VOLUME, MEAN $\pm$ SD*		LAST-2-SEC VOLUME, MEAN $\pm$ SD*		FORCED EXPIR. TIME, MEAN $\pm$ SD*	
	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994	1993	1994
Alpine (1)	5.92	7.00	72.2	78.0	43.2	61.3	59.4	77.7	4.7 $\pm 6.4$	3.7 $\pm 1.8$	61 $\pm 302$	8 $\pm 50$	6364 $\pm 1989$	6723 $\pm 1464$
L.Els. (2)	5.82	7.00	67.1	83.9	44.8	64.3	65.9	76.2	4.1 $\pm 2.5$	3.7 $\pm 1.9$	34 $\pm 276$	13 $\pm 113$	6521 $\pm 1736$	6706 $\pm 1689$
L.Gre. (3)	5.86	6.96	68.4	80.6	44.5	62.6	64.5	77.1	4.1 $\pm 2.1$	3.7 $\pm 1.7$	35 $\pm 202$	10 $\pm 42$	6311 $\pm 1606$	6265 $\pm 1442$
Lanc. (4)	5.64	6.98	71.4	80.6	49.0	59.9	68.5	73.6	4.0 $\pm 1.8$	3.8 $\pm 2.0$	27 $\pm 159$	17 $\pm 131$	6410 $\pm 1721$	6383 $\pm 1610$
Lomp. (5)	5.68	7.00	72.9	81.9	49.3	66.0	66.9	79.9	4.0 $\pm 1.8$	3.6 $\pm 1.7$	39 $\pm 351$	10 $\pm 48$	6544 $\pm 1766$	6378 $\pm 1375$
Lg.B. (6)	5.76	7.00	70.5	77.5	46.6	60.6	65.8	76.5	4.3 $\pm 2.0$	3.9 $\pm 2.0$	40 $\pm 244$	4 $\pm 32$	6796 $\pm 2024$	6667 $\pm 1563$
M.Lo. (7)	6.04	6.96	67.4	82.2	41.6	61.5	61.3	74.0	4.4 $\pm 2.1$	3.9 $\pm 1.9$	46 $\pm 256$	11 $\pm 85$	6560 $\pm 1841$	6919 $\pm 1693$
S.Dm. (8)	6.04	6.96	67.1	77.6	41.9	57.0	61.8	72.8	4.4 $\pm 2.1$	4.1 $\pm 2.1$	42 $\pm 263$	9 $\pm 59$	6845 $\pm 1880$	6684 $\pm 1731$
Atas. (9)	6.00	6.92	70.7	78.9	43.4	61.4	60.6	76.4	4.4 $\pm 2.1$	3.7 $\pm 1.6$	42 $\pm 248$	11 $\pm 61$	6365 $\pm 1747$	6699 $\pm 1586$
S.Mar. (10)	5.32	6.98	78.3	81.4	54.4	64.4	69.3	78.4	3.9 $\pm 2.0$	3.7 $\pm 1.7$	33 $\pm 182$	7 $\pm 56$	6325 $\pm 1634$	6467 $\pm 1373$
Upld. (11)	5.92	6.98	66.8	81.2	42.4	61.1	63.0	74.5	4.2 $\pm 2.2$	3.9 $\pm 1.9$	33 $\pm 195$	18 $\pm 194$	6722 $\pm 1971$	6745 $\pm 1602$
Riv. (12)	5.72	7.00	69.4	82.7	45.8	65.4	65.5	78.5	4.1 $\pm 1.8$	3.7 $\pm 1.5$	56 $\pm 354$	14 $\pm 86$	6330 $\pm 1680$	6428 $\pm 1498$
ALL	5.84	6.98	69.8	80.5	45.2	62.0	64.0	76.2	4.2 $\pm 2.7$	3.8 $\pm 1.8$	41 $\pm 260$	11 $\pm 90$	6512 $\pm 1817$	6595 $\pm 1574$

\*Considering technician-accepted blows only.

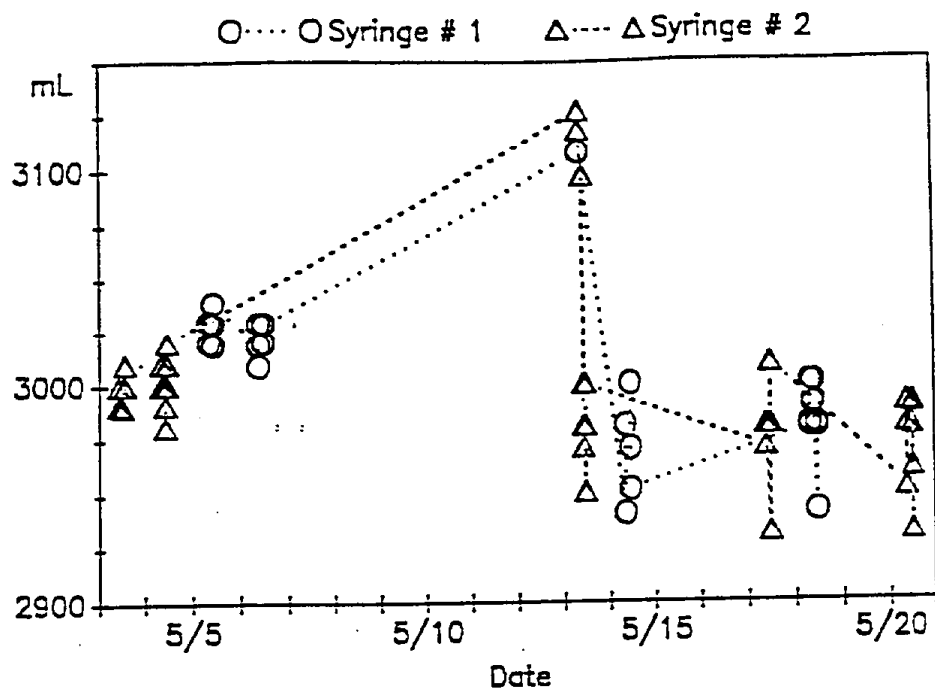
FEV<sub>1</sub> extrapolation volume in percent, last-2-seconds volume in ml, forced expiratory time in milliseconds. American Thoracic Society Guidelines: extrapolation volume < 5.0%, last-2-seconds volume < 200 ml, forced expiratory time > 6000 msec.

Figure 5.2.1



**FIGURE 5.2.2**

**CALIBRATION CHECK FVC READINGS  
FOR SPIROMETER #1**



**CALIBRATION CHECK FVC READINGS  
FOR SPIROMETER #4**

